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FAT UTILIZATION IN SUBMAXIMAL EXERCISE AS DETERMINED BY RESPIRATORY MEASURES.

University — Université

UNIV. OF ALBERTA

Degree for which thesis was presented — Grade pour lequel cette thèse fut présentée

M. Sc.

Year this degree conferred — Année d'obtention de ce grade	Name of Supervisor — Nom du directeur de thèse
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FAT UTILIZATION IN SUBMAXIMAL EXERCISE AS DETERMINED BY RESPIRATORY MEASURES

by

© DAVID LORNE MANN

A THESIS

SUBMITTED TO THE FACULTY OF GRADUATE STUDIES

AND RESEARCH IN PARTIAL FULFILLMENT OF THE

REQUIREMENTS FOR THE DEGREE OF MASTER OF SCIENCE

DEPARTMENT OF PHYSICAL EDUCATION

EDMONTON, ALBERTA

SPRING 1982

THE UNIVERSITY OF ALBERTA

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ABSTRACT

The relative contribution of various substrates to metabolism, as determined by respiratory measures, was measured for 12 male subjects ($\bar{x}$ age = 23.3 years) working at submaximal intensities. The subjects were divided equally, according to $\dot{V}O_2max$, into a trained (T) group ($\bar{x}$ $\dot{V}O_2max$ = 50.5 ml/kg/min) and an untrained (UNT) group ($\bar{x}$ $\dot{V}O_2max$ = 42.1 ml/kg/min). Each subject completed one session of each of 30, 40, 50 and 60% $\dot{V}O_2max$ exercise on a bicycle ergometer. A session included phases of rest, 10 minutes warm-up, 40 minutes exercise and 30 minutes recovery. Other measures taken included heart rate, lactate levels, urine urea N_2, diet analysis and anaerobic threshold determination. The dependent variable was a modified non-protein RQ (npR').

The npR' value was significantly different across phases. The highest values were recorded during exercise, followed by those of warm-up, then those of recovery. Recovery npR' did not decline below resting levels as observed elsewhere. Exercise intensity dictated the npR' value but had no effect on recovery npR', perhaps due to an insufficient stimulus. Finally, an indirect determination of the optimal intensity for fat utilization was made (40-50% $\dot{V}O_2max$). It was concluded that if an individual adhered to the lower ranges of a cardiovascular fitness program, he/she may also benefit from enhanced fat utilization.

ACKNOWLEDGEMENTS

The author would like to extend his gratitude to his advisor, Dr. Art Quinney, for his valuable guidance and encouragement throughout the course of this study. The helpful advise from committee members Dr. W. Cottle and Mr. B. Nielsen was also appreciated. Also, thanks are extended to Cheryl Luchkow for her diligent typing and to the twelve subjects who donated their blood, sweat and fat to this study.

TABLE OF CONTENTS

CHAPTER		PAGE
I.	INTRODUCTION	1
II.	REVIEW OF THE LITERATURE	4
	Substrate Metabolism	4
	Fat mobilization	5
	Factors controlling fat mobilizaton and oxidation	6
	Training effects	9
	Anaerobic threshold	11
	Use of Respiratory Measures in Determining Substrate Contribution	13
	Historical backgroud	13
	Procedures, validation and results	16
	Protein utilization	17
	Limitations	19
III.	METHODOLOGY	20
	Subjects	20
	Procedures	20
	Recent physical activity	21
	Body weight	21
	Resting blood pressure	21
	Submaximal predictive test	21
	$\dot{V}O_2max$	22
	HRmax	22
	Aerobic and anaerobic thresholds	23

CHAPTER PAGE

$\dot{V}O_2$ and $\dot{V}CO_2$ during rest and submaximal exercise . . . 23

HR during rest and submaximal exercise 23

Venous lactate levels 24

Urine urea N_2 24

Percent body fat 25

Percentage carbohydrate, fat and protein intake . . . 25

Experimental Design 25

Calculations 26

Statistical Analysis 27

IV. RESULTS AND DISCUSSION 28

Results 28

Physical characteristics 28

Diet analysis 29

Protein catabolism 29

Work capacity measurements 30

Exercise response measurements 31

Covariate treatment 35

Optimal exercise intensity 39

Discussion 39

Initial factors 39

Aerobic and anaerobic thresholds 41

Work intensity 41

Non-protein R' 44

Substrate contribution 44

Optimal exercise intensity 48

V. SUMMARY AND CONCLUSIONS 52

***

PAGE

REFERENCES 54

APPENDICES

APPENDIX A PHYSICAL ACTIVITY READINESS QUESTIONNAIRE (PAR-Q) . . . 62

APPENDIX B INFORMED CONSENT FORM 64

APPENDIX C CALCULATIONS COMPLETED BY THE BECKMAN METABOLIC CART . . 66

APPENDIX D GRAPHICAL DETERMINATION OF AEROBIC AND ANAEROBIC THRESHOLDS 69

APPENDIX E LACTIC ACID DETERMINATION 72

APPENDIX F UREA NITROGEN DETERMINATION 74

APPENDIX G CALCULATION OF PERCENT BODY FAT 76

APPENDIX H SAMPLE DIET ANALYSIS 78

APPENDIX I CALCULATIONS FOR DETERMINING npR' 86

APPENDIX J RAW DATA 88

APPENDIX K REGRESSION PROCEDURES FOR COVARIATE DETERMINATION . . . 96

APPENDIX L ANOVA TABLES 98

APPENDIX M INDIRECT DETERMINATION OF OPTIMAL EXERCISE INTENSITY . . 103

LIST OF TABLES

Table	Description	Page
4.1	Physical Characteristics of the Subjects	28
4.2	Diet Analysis	29
4.3	Work Capacity Measurements	30
4.4	Absolute AerT and AnT Measures	30
4.5	Heart Rates	33
4.6	Lactic Acid	33
4.7	Non-Protein R' Values	34
4.8	Summarized Post-Hoc Comparisons From npR' ANOVA	38
4.9	Kilocalories Expended From Fat Sources	40
4.10	Comparison of UNT AerT and AnT to Those Reported in the Literature	43

LIST OF FIGURES

Figure		Page
4.1	Changes in npR' For Both Pooled (+) and Group (*) Data as a Function of Phase	36
4.2	Changes in Pooled npR' as a Function of Intensity and Phase	37
4.3	Kilocalorie Expenditure From Fat Sources For T (*) and UNT (+) Subjects as a Function of Regression-Generated Intensity	49

CHAPTER I

INTRODUCTION

In recent years there has been considerable emphasis on physical fitness in our society. One aspect of physical fitness is the relative proportion of fat stored in the body. A fitness and health concern is the common excess of body fat in a large segment of our population. Good physical fitness has been defined as the minimal acceptable levels of strength, flexibility, cardiovascular endurance, and body composition necessary to maintain adequate health (Astrand and Rodahl, 1977). A common means of reducing body fat is to increase activity levels. Research has indicated that there are intensities at which fat is preferentially utilized (i.e., Pirnay et al., 1977). Studies of substance utilization have shown that fat is the predominate fuel at the low end of the exercise intensity continuum. Conversely, carbohydrates (CHO) become predominate as intensity increases. As excess body weight can be related to excess fat storage, it is of interest to determine how best to 'burn' this excess off. Because slower, longer activity both taxes the cardiovascular system as well as oxidizing predominately fats, a light prolonged exercise protocol will best elucidate the question of optimal fat utilization.

There are several ways to measure substrate utilization in light, prolonged exercise. One method is to take serial blood samples. From these samples numerous substances can be identified: glucose, lactate, insulin, free fatty acids (FFA), free glycerol, catecholamines, glucagon, etc. Each of the substances, when analysed serially, are indicative of a component of the process of substrate utilization. One difficulty with such a technique is that the substances are blood borne and therefore

are only in transit. Although meaningful indications of rates of flows and the onset times of these flows can be made, conclusions about the cellular processes are not possible. To get at this problem the investigator must look at more microscopic techniques such as biopsy and in vitro experimentation. Such studies have provided valuable knowledge to this topic. However, with increasing experimental sophistication comes increasing technical difficulties. Serial blood analyses require numerous samplings, sometimes elaborate assays and the trained personnel and proper laboratory facilities for such processes. Biopsies, an invasive technique, are equally technically difficult. Thus, other methods were sought that were reliable yet more easily administered.

The common method of energy expenditure and substrate utilization determination is to use respiratory measures. The principle is that the oxidation of different substrates produces different ratios of CO_2 produced to O_2 consumed. It has also been determined that the consumption of a litre of O_2 at a given ratio is directly associated with the expenditure of given amounts of energy. Thus, within limitations, respiratory measures can be used to obtain energy and substrate data. The advantage of the method is that it is non-invasive, reasonably reliable under certain conditions, and can be administered easily.

The purpose of this study was to monitor substrate utilization, as measured by respiratory parameters, during submaximal exercise. The exercise protocol was designed to approximate a 1 hour session that a sedentary person might undertake in a fitness program. While the work phase lasted 50 minutes, resting and recovery measures were also recorded. Various factors that may influence the fat contribution profile - such as diet, $\dot{V}O_2max$, anaerobic thresholds, protein catabolism and lactate

levels - were also measured. The ultimate goal was to determine an optimal intensity for fat utilization and compare this to established fitness protocols.

Several limitations affected the findings of the study. Besides the usual difficulties involved with data collection and analysis, problems arose concerning the determination of work intensities. Also, the fact that respiratory measures are indirect assessors of substrate contribution must be taken into consideration when interpreting the results.

CHAPTER II

REVIEW OF THE LITERATURE

Substrate Metabolism

It has been known for many years that three principle foodstuffs (fats, proteins and carbohydrates [CHO]) are used in energy production by the human body (Regnault and Reist, 1849). Early investigations further determined that the contribution of proteins is small and can be quantified through fecal and urine samples (Zuntz and Schumberg, 1901; Lusk, 1928; Cathcart and Cuthbertson, 1931). This has left only fats and CHO as the principle contributors to the energy obtained either at rest or during various modes and intensities of exercise.

Substrate contribution over an extended submaximal exercise bout takes on a characteristic profile. Generally, as lower intensity exercise continues over a long period (i.e., greater than 30 min), the fat contribution increases while the CHO contribution decreases (Benadé et al., 1971; Girandola and Katch, 1976; Pirnay et al., 1977). This is true for intensities where the inhibition to fat mobilization does not exceed its stimulation; usually not greater than 70% $\dot{V}O_2$max (Pruett, 1970).

The means of physical warm-up may alter the substrate profile. Borysyk (1977) compared two modes of warm-up - exercise at 40% $\dot{V}O_2$max (T_e) and sauna (T_s) - with no warm-up (T_c). The results showed elevated lactate levels under T_s and T_c conditions, suggesting a greater CHO contribution in the absence of an exercise (T_e) warm-up.

The recovery phase also presents some interesting characteristics. Pruett (1970), using well trained males, found plasma free fatty acids (FFA) to be elevated several hours after a near maximal exercise bout.

During the actual exercise plasma FFA levels were depressed, as expected, but rose dramatically at the onset of recovery. Also, a significant plasma FFA a-v difference was noted indicating fat utilization in recovery. Farley and Hamley (1977) have reported that the respiratory quotient (RQ) remains low after exercise, further suggesting fat utilization during recovery.

Other factors may also change this profile. Carbohydrate ingestion during exercise will cause, after a latency period of about 10 minutes, increases in blood glucose, insulin and pyruvate (Benadé et al., 1971; Benadé et al., 1973a; Girandola and Katch, 1976; Pirnay et al., 1977). These findings suggest a relatively greater CHO contribution, a suggestion verified by Benadé and co-workers (1973a) observations of an increased RQ following sucrose ingestion. Also, subjects ingesting sucrose in recovery display drastic declines in plasma FFA levels (Pruett, 1970).

Diet may have profound effects on the substrate contribution profile as measured by RQ. Increasing the dietary content of fats or CHO will cause a concomitant rise in the energy contribution of that substrate in exercise (Issekutz et al., 1963; Bergstrom et al., 1967; Maughan et al., 1978); however Issekutz and co-workers (1963) suggested that the CHO content had the greatest effect on the RQ value. Also, the higher the dietary fat content, the sooner the onset of exhaustion; a situation reversed by CHO feeding (Bergstrom et al., 1967; Brooke and Green, 1975).

1. Fat mobilization

Fats are stored as triglycerides (TG) both in adipose tissue and within the muscle itself (Gollnick, 1978). The release of endogenous TG for muscle use is a simple mass action effect, with the oxidative fibers (Type I) having greater TG stores (Essen, 1978). However, the mobilization

of fatty acids (FA) from adipocytes to blood borne FFA constitutes several steps.

The mobilization of FFA (lipolysis) is principally mediated by a hormone-sensitive lipase (HSL) reaction. The process is initiated when a given hormone attaches to the receptor site on the adipocyte. Near the site adenylate cyclase is activated to produce c-AMP, which in turn activates a protein kinase that converts the inactive lipase to the active form (Steinberg, 1976). There are, in fact, three lipases, each necessary to break TG down to glycerol and three FA (Steinberg, 1976).

Once the FA is released from the stored TG it moves across the membrane to be bound to circulating albumin; the uptake of FA by albumin being dependent on albumin concentration (Spector and Fletcher, 1978). The FA (now called FFA) is carried by albumin to the target cell where it is released, again down a concentration gradient into the cell (Pande, 1971). Fatty substances are also circulating in the esterfied form, such as lipoprotein complexes and chylomicrons. The energy contribution from these sources is thought to be low (Mackie et al., 1980).

While the FFA may pass through the cellular membrane passively, the molecule must be actively moved into the mitochondria. This requires a FFA shuttle in the presence of ATP, thiokinase and carnitine (Pande, 1971). Once in the mitochondria the FFA is in the acetyl-CoA form in which it enters the β-oxidation process. This process removes acetyl-CoA groups which ultimately enter Krebs' cycle for the production of ATP.

2. Factors controlling fat mobilization and oxidation

At the adipocyte several factors control the mobilization of FFA. The principal means of mobilization is via the HSL system. Although many hormones have been identified that stimulate lipolysis, of particular

interest to exercise is the epinephrine and nonepinephrine effect. These catecholamines cause a sudden acceleration in lipolysis (Fain et al., 1978) that may be 10 - 15 times basal rate under highly stressful conditions (Guyton, 1976). Although the mechanisms are not clear, Burns and co-workers (1975) have suggested that the hormones act at the adenylate cyclase level. Sympathetic nerve endings also release catecholamines (Ganong, 1979) and these terminals are present in adipose tissue (Newsholme and Start, 1976). Sembrowich and co-workers (1974) monitored rat lipolysis during exercise under surgical adrenomedullectomy, chemical sympathectomy, or a combination of both. They found that lipolytic stimulation from the adrenals and the adrenergic nerves were additive, with the nervous influence being the predominate factor. It was also noted that total removal of the adrenergic system did not completely stop exercise-induced lipolysis, suggesting some other factor contributes to lipolysis. The mass action effect due to available albumin (Spector and Fletcher, 1978) may be a contributor.

Other hormones have been identified as lipolytic agents. These include thyroid hormones (TH), growth hormone (GH), glucocorticoids (GC), cortisol and adrenocorticotropic hormone (ACTH). The TH, GH and GC appear to have a direct effect on lipolysis while ACTH and cortisol effects are mediated through GC production (Tharp, 1975). These hormones chronically raise lipolysis activity as opposed to the more acute catecholamine effect (Fain et al., 1978). The final hormone of importance is insulin, which has antilipolytic effects. Insulin promotes the movement of glucose into the cell where it is synthesized to α- glycerophosphate which in turn binds with intracellular FA to form TG (Gollnick and King, 1969). Insulin also stimulates LPL release from capillary endothelial cells (Pruett, 1970) which will promote fat storage. Finally, the hormone has been shown

to inhibit adipocytic c-AMP (Froesch, 1967), thus reducing lipolysis.

Another important regulating factor at the adipocyte is the apparent inhibition of lipolysis by lactate (Boyd et al., 1974; Gollnick and King, 1973; Havel et al., 1967; Holloszy, 1973; Paul and Holmes, 1975; Pruett, 1970; Wenger and Reed, 1976). Two of these groups (Gollnick and King, 1973; Paul and Holmes, 1975) further suggested that lactate may stimulate fat storage via α- glycerophosphate production. Havel and co-workers (1967) believe that the inhibition occurs at the c-AMP level. Therefore, these are two regulatory systems operating on the lipolysis reaction during exercise. Lipolysis is enhanced by a mass action effect plus catecholamine stimulation while the process is retarded by lactate accumulation. Pruett (1970) has suggested that at 70-80% of $\dot{V}O_2max$ optimum FFA mobilization occurs, although she was dealing with highly trained endurance athletes.

In the muscle cell itself the fat used for energy production is dependent on many factors. In the FFA shuttle system to the mitochondria, the shuttle enzyme thiokinase is more active in oxidative fibers (Pande, 1971), probably due to higher mitochondrial density. The enzyme is also sensitive to energy charge and high concentrations of ADP and acyl-CoA, products of the shuttle system (Pande, 1971). Once inside the mitochondria, the β-oxidation process also displays rate-limiting steps. These occur with the varying re-dox nucleotide ratios which are oxygen supply sensitive (Hochachka, 1977). Thus, oxygen deficiency may reduce the efficiency of the shuttle. A buildup of anaerobic by-products (such as ADP, H^+, etc.) may also shift the energy charge unfavourably for thiokinase activity.

The regulation mechanisms in Krebs' cycle involve interaction with glucose metabolism. Fat oxidation will tend to have a glucose sparing effect (Armstrong, 1976; Essen, 1978; Newsholme, 1978; Wenger and Reed,

1976). This means that if energy metabolism is directed through the fat oxidative pathway, rather than the glycolytic pathway, the consequence of the fatty acid oxidation will be to further inhibit glucose utilization. This is accomplished via two main inhibitory mechanisms on glycolysis. High concentrations if citrate, a Krebs' cycle by-product, will inhibit phosphofructokinase (PFK) (Newsholme, 1978). PFK is a rate limiting enzyme in glycolysis. The other factor is that high acetyl-CoA/CoA ratios inhibit pyruvate oxidation (Wenger and Reed, 1976), again slowing aerobic glycolysis. This glycose sparing effect may be reduced as work intensities increase and the fat oxidative system is not able to meet the energy demands. In this situation, PFK activating substances (low energy charge, P_i, NH_4^+) will accumulate, causing an increased flux through glycolysis (Newsholme, 1978). With the falling energy charge also reducing thiokinase activity, and increased lactate lowering fat mobilization, acetyl CoA/CoA ratios might be expected to fall, thereby removing its inhibition on pyruvate oxidation and further promoting glycolysis.

3. Training effects

Recent work has been done on the effect of training on rat lipolysis. In general, endurance training has shown increases in epinephrine stimulated lipolysis (ESL) in vitro (Askew et al., 1975; Howle and Barnard, 1976; Askew and Hecker, 1976; Owens et al., 1977). However, the exact mechanism has not been determined (Askew et al., 1975). Earlier studies had not controlled for the change in adipocyte size observed with training, although later work (i.e., Askew and Hecker, 1976) estimated ESL relative to cell size and still found significant training effects. Investigations of the lipolytic mechanisms have thus far found no changes. Several authors (McGarr et al., 1976; Shephard et al., 1977; Oscai, 1979) have found

no increase in HSL with training, although HSL activity remains elevated after chronic (McGarr et al., 1976) and acute exercise (Oscai, 1979). A group of investigators (Oscai et al., 1981; Palmer et al., 1981) recently examined the training effect on HSL precursors. They found increased c-AMP activity but decreased protein kinase concentrations with no change in HSL activity. They concluded that training does not enhance lipolysis, suggesting that some other mechanism is responsible for the observed ESL training effect in rats. However, this conclusion is not supported by Shepherd and co-workers (1981) who state that training does enhance lipolysis. Their reasoning is that the HSL precursors may not be the sole controlling point of HSL, and subsequently lipolysis. They further proposed the presence of a c-AMP independent protein kinase as suggested elsewhere (Schlender and Reimann, 1975) as well as an altered HSL sensitivity due to training. More work will be needed in this area to elucidate these differences of opinion.

Considerable work has been done investigating the effects of endurance training on muscle cell metabolism. Generally, endurance training will enhance selective fat metabolism enzymes associated with transport and cyclic oxidation (Bengi et al., 1975; Gollnick and King, 1969; Holloszy, 1973; Holloszy, 1975; Molé et al., 1971). Trained individuals also display lower absolute lactate levels at equal workloads (Astrand and Rodahl, 1977; Bransford and Howley, 1979). Coupled with cardiovascular adaptations (Astrand and Rodahl, 1977), these metabolic/enzymatic adaptations will enhance a trained individual's aerobic capacity. This will result in greater utilization of fat as a fuel.

Research into the training effects on resting and sub-maximal plasma FFA have revealed lower plasma FFA levels in trained rats (Winder et al., 1975) and humans (Johnson and Walton, 1972; Johnson et al., 1969; Bransford

and Howley, 1979; Wirth et al., 1979). The explanation for such findings are either reduced mobilization or enhanced uptake and oxidation. In view of the previous discussion on training-stimulated ESL and enhanced cardiovascular and cellular aerobic capacity, it would appear that the latter explanation is more likely. As further evidence, Wirth and co-workers (1979) agree with this alternative; citing their finding that, while training decreased plasma FFA, plasma glycerol remained unchanged. Also Girandola and Katch (1976) reported that training lowered submaximal RQ, indicating greater fat oxidation.

4. Anaerobic threshold

The onset of anaerobic metabolism is significant to substrate contribution because of lactate production and fuel shifts. In the last ten years considerable work has been done in measuring and explaining the onset of anaerobiosis. By definition, the anaerobic threshold (AnT) is the "... level of work or O_2 consumption just below that at which metabolic acidosis and associated changes in gas exchange occur" (Wasserman, et al., 1973). Metabolic acidosis is imperically defined as lactic acid production of greater than 4 mmoles/l (Kinderman et al., 1979) while changed gas exchange values include $\dot{V}_e$, $\dot{V}CO_2$, F_eO_2, F_eCO_2 and R (Skinner and McLellan, 1980). The R value has been found to be unreliable as a threshold indicator (Davis et al., 1976).

Recently Skinner and McLellan (1980) presented an aerobic-anaerobic transition model based on the results found in the literature. In particular, these authors noted that reported anaerobic thresholds range from 40-80% of $\dot{V}O_2$max. Under closer examination it was suggested that two transitions or thresholds occur; the first in the 40-60% range and the second in the 60-80% range. These were named the aerobic (AerT)

and anaerobic threshold (AnT), respectively. The AerT is defined by a minimum F_eO_2 value and discontinuous increases in $\dot{V}_e$ and $\dot{V}CO_2$ plus a rise in blood lactate levels to about 2 mmoles/l. The AnT is signalled by a maximum F_eCO_2 value, a further discontinuous increase in $\dot{V}_e$ and blood lactate levels of 4 mmoles/l. The occurrence of two thresholds necessitates the formation of three phases known, respectively, as Phase I, II and III. As the individual works through these phases, Skinner and McLellan (1980) suggest that the fuel source shifts from primarily fats in Phase I to primarily CHO in Phase III.

The hypothetical model developed by Skinner and McLellan (1980) is meaningful in light of earlier work. Some previous studies (i.e., Weltman et al., 1978) reported anaerobic thresholds of approximately 50% $\dot{V}O_2$max while others (i.e., Kinderman et al., 1979) report thresholds of 80% $\dot{V}O_2$max. When the Skinner-McLennan two-threshold model is applied to these data the results are more easily explained.

Davis and co-workers (1976) investigated the inter-correlations between the various threshold indices. They were able to show a strong relationship between gas exchange parameters and the more objective criteria of blood lactate ($r = .95$). The test-retest results were not as high ($r \simeq .75$) although others (Davis et al., 1979) obtained correlations of $r = .91$.

Anaerobic threshold appears to respond to a training stimulus. Davis and co-workers (1979) were able to show absolute increases of 44% and relative (to $\dot{V}O_2$max) increases of 15%. It has also been noted that endurance athletes with similar $\dot{V}O_2$max values may perform quite differently (Costill et al., 1973) and that an athlete may reach a peak $\dot{V}O_2$max yet still continue to improve performance times (Astrand and Rodahl, 1977, p. 413). These observations may be explained in part by a training-

dependent threshold. Individuals with a higher threshold would be expected to utilize more fat at a given relative workload (Davis et al., 1979; Stamford et al., 1978; Weltman et al., 1978) and expend less total energy (Weltman et al., 1978).

Use of Respiratory Measures in Determining Substrate Contribution

1. Historical background

Perhaps the earliest related experiments were conducted by Lavoiser when he noted that any volume of O_2 combined with carbon produced the same volume of CO_2. However, he also found that the volume of inspired O_2 was greater than the volume of expired CO_2, concluding that the excess O_2 must have been used to oxidize hydrogen to water (Lusk, 1928). This ratio of gases expired to inspired was called the respiratory quotient or RQ (Pflüger as reported by Lusk, 1928). Regnault and Reiset in 1849 were the first to demonstrate that the RQ was dependent on diet (Lusk, 1928). These experiments were done on fowl and dogs. By the late 1800's and early 1900's investigators were gathering data to formulate tables based on the RQ. It was known at this time that three main foodstuffs contribute to energy generation - proteins, fats and carbohydrates - and that the RQ is indicative of the relative combustion of these fuels. As the contribution of protein was small and could be estimated from fecal and urinary excretion, the RQ tables became non-protein RQ tables by subtracting out the protein contribution (usually via urinary analysis) from the total measured CO_2 production. The first such table was produced by Zuntz and Schumburg (1901). This table was standard until 1924 when Lusk published slightly corrected values. The Zuntz-Schumburg-Lusk tables were shown to be erroneous by Cathcart and Cuthbertson in 1931. These authors pointed out that the earlier work had been based on in vitro

studies of adipose tissue while blood, muscle, and liver lipids had been ignored. Having made the necessary corrections, these tables have been standard since that time. It must be noted that the revisions to these tables have resulted in relatively minor changes, such that preferential use of the Zuntz-Schumburg-Lusk or Cathcart-Cuthbertson tables makes little difference.

Several attempts have also been made to estimate other physiological phenomena from the RQ. In the early 1960's Issekutz and co-workers did a series of experiments measuring respiratory factors. Issekutz et al. (1962) attempted to assess aerobic work capacity with the RQ. Using a modified RQ the authors found the measure a reasonably good predictor of aerobic power, especially at RQ values greater than unity. However, the advantage of using R values instead of oxygen uptake was not explained. Issekutz and Rodahl (1961) also investigated the RQ under short, high intensity work to exhaustion. They concluded that the RQ may indicate the percentile participation of the anaerobic glycolysis system as, under these conditions, the measure is dependent on O_2 supply and blood acid levels. Finally, Wasserman et al. (1973) evaluated the RQ as an indicator of anaerobic threshold. However, they concluded the measure to be the least sensitive of all the threshold determination indicators. It appears that the measurement of the RQ is best suited for calorific investigations.

More recently the term respiratory exchange ratio (RER), rather than the RQ, has been used as the measure of the ratio of expired CO_2 to inspired O_2 at the mouth. This review will not attempt to settle this difference. As a compromise the initial 'R' will be referred to as the measure used to determine substrate utilization. This application has been used in the recent literature (i.e., Bursztein et al., 1980).

2. Procedures, validation, and results

As has already been discussed, the basic procedures have been with us for many decades. After Cathcart and Cuthbertson (1931) established their standard tables, Weir (1949) published a technique to determine the non-protein R without urinary protein analysis. He was able to show that the error due to protein, while significant, is relatively constant under conditions of stable diet. The estimate was that approximately 12.5% of total energy expenditure can be related to protein. Thus, by introducing a mathematical constant in place of urinary analysis, Weir has simplified the technique. Kleiber (1965), however, states that while Weir's formula might simplify the procedure it does not add to the knowledge of the metabolic processes.

Consolazio et al. (1963) present a format for the determination of protein, fat, and CHO catabolized. This procedure requires the determination of the non-protein R via urinary analysis. These same authors discussed Weir's method at length; including comparing it to conventional methods. It was suggested that if corrections were made for an individual's daily protein intake, the "... Weir formula ... makes no significant change in kilocalorie expenditure" (p. 324). From this point the non-protein R can be computed and substrate contribution determined. It must be noted that the above procedures are usually employed at rest. It can be seen that substrate contribution can be quite accurately determined at rest but exercising results in shifts in respiratory functions, particularly the expired CO_2.

With the onset of anaerobiosis there will be seen a concomitant increase in blood lactate (Astrand and Rodahl, 1977). This buildup will require buffering to maintain blood pH. The results of this buffering action will be an excessive production of blood CO_2, subsequently being

'blown off' at the lungs. Such excess CO_2 would confound R and mask the substrate effect on the measure. Thus, respiratory substrate studies cannot be done reliably at intensities which might elicit this excess CO_2. Several researchers have pointed this out (Garby and Lammert, 1977; Issekutz et al., 1963; Issekutz et al., 1964). Christensen and Hansen (1939a) suggested that the R measure is valid at high but not maximal work levels after 10-15 minutes.

Many authors have used R to estimate substrate utilization during exercise in humans (Brooke and Green, 1975; Garby and Lammert, 1977; Hermansen et al., 1967; Issekutz et al., 1963; Pirnay et al., 1977; Young et al., 1967). Benadé and co-workers (1971, 1973a,b) did a series of studies investigating substrate utilization during heavy physical work while Bursztein et al. (1980) used R to determine the metabolic state of critically ill patients. All the exercise studies reported here used non-protein R's with two groups measuring urinary protein (Issekutz et al., 1963; Pirnay et al., 1977) while the other groups did not. In the latter case protein excretion is assumed to be a constant, approximately 0.5 g/hr (Young et al., 1967), and the corresponding non-protein R determinations are made.

As respiratory measures are indirect indicators of substrate utilization, it is necessary to validate these measures against more direct methods. Consolazio et al. (1963) noted that early experiments showed that indirect calorimetry was in good agreement with direct methods in determining energy expenditure. With respect to the types of fuels expended, many investigators have found significant correlations between substrate depletion and R using biopsy analysis (Ahlborg et al., 1967; Bergstrom et al., 1967; Bergstrom et al., 1969; Hermansen et al, 1967). Benade et al. (1973)b induced labelled carbon in the diet and

found a good correlation between substrate contribution as measured by R and labelled CO_2. These authors also addressed themselves to the question of whether the shifts seen in R are related to substrate metabolism or some confounding substance (i.e., insulin). The conclusion was that the changes are associated with shifts in the glycolysis/fat oxidative system and that R is a valid measure of the conversion of pyruvate to CO_2 under submaximal conditions. Studies have been conducted based upon these and the biopsy conclusions. Benadé et al. (1971) studied substrate utilization over 6 hours based on these premises. Girandola and Katch (1976) state that they "... believe steady-state R values are a reliable estimate of fuel utilization" (p. 123). Finally, Turell and Alexander (1964) compared three methods of metabolic rate determination: Weir's formula, Zuntz-Schumburg-Lusk tables, and Cathcart and Cuthbertson tables. They found that all three methods were within 1% of each other. Their conclusions were that any of these methods are valid in the resting or steady-state condition. It appears that respiratory measures, when used under the proper conditions and observing the necessary correction factors, are a valid method for estimation of substrate utilization.

3. Protein utilization

As protein constitutes one of the three major foodstuffs, it is necessary to discuss its role in the measurement of substrate utilization. The literature shows that there is no consensus on the importance of protein as a fuel in light exercise. For example, Castenfors and Piscator (1967) report slight but non-significant rises in urinary protein with light exercise. Margaria and Foa (1939) suggested that protein plays a non-essential role while Bursztein et al. (1977) state that error in not

measuring protein decreases with increasing metabolic rate. Nevertheless, Lemon and Mullin (1980), in studying protein excretion via blood and sweat as well as urine, concluded that "... protein is utilized to a greater extent than realized" (p. 624).

The usual method of protein determination for non-protein R values, as has already been mentioned, is with urinary urea analysis. Such procedures are thoroughly described in Kleiber (1965) and Consolazio et al. (1963). The procedure is to determine the amount of urea nitrogen (gms) produced and multiply this figure by 6.25 (the computed gram equivalent) to obtain the amount of protein utilized. As this figure is expressed in grams, the gram equivalent contributions of fats and CHO are also computed - resulting in a percentile contribution of all three substances.

The above procedures have been used by several investigators (Brooke and Green, 1975; Bursztein et al., 1980; Issekutz et al., 1963; Pirnay et al., 1977). Pirnay and co-workers reported that protein provides 1-2% of the energy expended in submaximal treadmill work, while the other investigators did not even report protein percentiles. Young et al. (1967) went one step further and assumed a constant urinary N_2 excretion rate. Thus, to this point it appears that protein does assume the non-essential role as stated by Margaria and Foa (1939). However, the recent work of Lemon and Mullin (1980) does not support this assumption. In measuring urea excretion rates in serum, sweat and urine in one hour of 60% $\dot{V}O_2$max work under conditions of CHO loading and depletion. These authors found that protein contributed 10.4% of the energy expended under the CHO depleted condition. While an increase in protein contribution was also seen under the CHO loaded condition, the contribution was not nearly so high. Lemon and Mullin suggest that protein catabolism values

may be underestimated due to three main reasons:

a) urea N_2 does not account for 100% of the protein catabolized (only 80-90%)

b) 15-30% of the urea is hydrolysed in the alimentary tract

c) urea excretion may remain above resting levels for up to 4 hours at the end of exercise.

4. Limitations

As with any biological measure, there are certain limitations that must be observed so that a true measure might be obtained. Such is the case with respiratory measures for determining substrate utilization. Consolazio and co-authors (1963) have outlined some of these. The first and most important point is to be sure that R is truly representative of the combustion mixture. This is not possible under the conditions of acidosis or alkalosis, which can occur during non-steady-state exercise. Acidosis is a particular problem with the onset of anaerobiosis. For this reason exercise intensity should be maintained below about 50% of $\dot{V}O_2max$, which is approximately the point at which blood lactate levels begin to increase (Astrand and Rodahl, 1977), although this can vary due to individual differences. This is verified by Issekutz and Rodahl (1961) in stating that the R measure can only be used in metabolic studies if blood CO_2 and lactate have reached steady-states, usually after 10-30 minutes, depending on the type of work. The other major limitation, as mentioned earlier, is diet. Strict control of pre-test diet is essential; usually accomplished by a prescribed diet and/or 12 hour fast prior to testing (Consolazio et al., 1963).

CHAPTER III

METHODOLOGY

Subjects

Twelve healthy males between the ages of 18 and 28 years ($\bar{x}$ = 23.3) volunteered to participate in the study. Subjects completed a PAR-Q and informed consent form (Appendices A and B, respectively) prior to testing. All subjects attended an orientation session, followed by a submaximal ergometer screening test for those with little experience in strenuous exercise (9 of 12). A maximal oxygen uptake test on the ergometer followed. A minimum of 3 days elapsed before the actual testing commenced. Four testing sessions were done in which each subject exercised at 30, 40, 50 and 60% of his $\dot{V}O_2$max. Participants were requested to maintain their regular activity pattern and diet throughout the testing period.

Subjects were selected to elicit either a high aerobic power (i.e., > 50 ml O_2/kg/min) or lower aerobic power (i.e., < 45 ml O_2/kg/min). The group with the higher value ($\bar{x}$ = 50.5 ml/kg/min) was designated trained (T) while the lower value group ($\bar{x}$ = 42.1 ml/kg/min) was designated untrained (UNT). Both groups had 6 subjects.

Procedures

The following measures were taken and recorded. Raw data appears under Appendix J.

- recent physical activity
- body weight (kg)
- resting blood pressure (mmHg)
- submaximal predictive test (ml/kg/min)
- $\dot{V}O_2$max (ml/kg/min), HRmax (BPM)
- anaerobic and aerobic thresholds (%$\dot{V}O_2$max)

- $\dot{V}O_2$ during rest, submaximal exercise and recovery (l/min, STPD)
- $\dot{V}CO_2$ during rest, submaximal exercise and recovery (l/min, STPD)
- HR during rest, submaximal exercise and recovery (BPM)
- venous blood lactate levels (mg %)
- urine urea nitrogen (g/hr)
- % body fat
- % carbohydrate intake
- % fat intake
- % protein intake

1. Recent physical activity

Subjects were asked to outline their regular physical activity over the past 1-2 years.

2. Body weight

Body weight was measured at the first testing session and recorded to the nearest 0.1 kg.

3. Resting blood pressure

Those subjects that stated that they were not used to vigorous physical exercise (9 of 12 subjects) had their resting blood pressure recorded. Participants with blood pressures greater than 150 mmHg systolic or 100 mmHg diastolic were to be excluded from the study. No subjects were excluded.

4. Submaximal predictive test

Subjects classified as inactive completed a submaximal predictive test. An Astrand-Rhyming (1954) six minute ergometer test was administered. Heart rate was measured at the end of each minute via a bi-polar cardiotachometer. Working at a constant rate of 60 RPM, workload was adjusted to elicit a heart rate between 125 and 170. Blood pressure was monitored each minute.

5. $\dot{V}O_2max$

A modified continuous incremental test, using a constant workload bicycle ergometer, was administered (Astrand and Rodahl, 1977). Rather than pedalling at 60 RPM, subjects selected their own rate; usually between 70 and 80 RPM. The intensity started at between 200 and 500 kpm/min, depending upon the training background and size of the subject. Workload was increased by 100 kpm/min approximately every minute, with the entire test lasting not more than 15 minutes. Heart rate was recorded every 30 seconds via a bi-polar cardiotachometer (Cardionics).

Maximum oxygen uptake ($\dot{V}O_2max$) was determined by an automated respiratory gas analysis apparatus (Metabolic Measurement Cart [MMC], Beckman Instruments). This system contains an OM-11 oxygen analyzer and a LB-2 carbon dioxide analyzer, as well as volume, temperature and pressure transducers. Data from these sensors and transducers were transmitted into the computer memory and transferred to the calculator for tabulation. During the $\dot{V}O_2max$ test expired gas samples were analyzed every 30 seconds. These included expired volume ($\dot{V}_E$, BTPS, l/min), fraction of expired O_2 and CO_2 (F_eO_2, F_eCO_2), respiratory exchange ratio (R), and oxygen consumption ($\dot{V}O_2$, STPD, l/min or ml/kg·min). These calculations appear in Appendix C. The metabolic cart was calibrated prior to each testing session.

The $\dot{V}O_2max$ value was determined by taking the highest value recorded. Subjects were verbally encouraged to continue until exhaustion. Two subjects displayed a leveling off of $\dot{V}O_2$ while all others showed distinctive increments in their last measure. Thus, the highest value usually corresponded to the last value obtained.

6. HRmax

Heart rate was recorded when the expired gases were recorded;

every 30 seconds of the $\dot{V}O_2$max test. HRmax was the highest attained heart rate, usually during the last 30 seconds of exercise.

7. Aerobic and anaerobic thresholds

Aerobic (AerT) and anaerobic (AnT) thresholds were determined graphically (Skinner and McLellan, 1980) from the data obtained during the $\dot{V}O_2$max test. Expired volume ($\dot{V}_e$), $\dot{V}CO_2$, F_eCO_2 and F_eO_2 were plotted against time. The AerT was defined as the disproportional increase in $\dot{V}CO_2$, the minimum F_eO_2 value and the first disproportional increase in $\dot{V}_e$. The AnT was defined as the maximum F_eCO_2 value and the second disproportional increase in $\dot{V}_e$. Threshold values determined by the various indices were averaged to give the reported AerT and AnT.

8. $\dot{V}O_2$ and $\dot{V}CO_2$ during rest and submaximal exercise

These values were generated by the MMC and expressed in litres per minute, STPD (see Appendix C). Resting and warm-up values were taken every minute and exercise and recovery values every 2 minutes. During rest the subject was asked to sit quietly with his eyes closed for 5 minutes or until 5 minutes of steady state was achieved.

9. HR during rest and submaximal exercise

Heart rate was monitored via bi-polar chest leads. Recordings were taken whenever the MMC measured an accumulated sample. Heart rate was averaged as follows:

- 5 minutes of rest
- 10 minutes of warm-up
- last 30 minutes of exercise
- first 10 minutes of recovery
- last 20 minutes of recovery

10. Venous lactate levels

Venous blood samples were taken from the anticubital vein. The sample was de-proteinated with 4% perchloric acid at 4°C. The de-proteinated sample was then stored at 4°C for no more than 90 minutes, at which time it was centrifuged at 3000 g's for 10 minutes. The supernatant was pipetted off and frozen at -57°C for later analysis. Analysis was done with the Sigma Chemical Co. (St. Louis, Missouri) Pyruvic Acid and Lactic Acid kit, which is based on a reversible enzymatic reaction (Henry, 1968). See Appendix E for details.

Samples were taken at rest, following 40 minutes of exercise and following 30 minutes of recovery. Approximately 3 mls of blood was taken at each sampling. Due to technical difficulties, blood sampling was not always successful. Subsequently not all lactate values were available.

11. Urine urea N_2

Protein catabolism was determined from urine urea N_2 levels and urine volume. Subjects were asked to void just prior to testing and immediately after recovery to provide an exercise urine sample. The void time and sample time were recorded. Three hundred millilitres of water was given prior to testing to assure a urine sample. The volume of this sample was measured and a 5 ml portion was frozen at -57°C for later analysis. Pilot analysis of frozen and fresh samples showed no difference, so all samples were frozen. Urea N_2 determination was done with the Sigma Chemical Co. (St. Louis, Missouri) Urine Urea Nitrogen kit. The procedure is based on the methods described by Fawcett and Scott (1960) and by Chaney and Marbach (1962); a chromogenic reaction between ammonia and phenol in the presence of sodium nitroprusside (see Appendix F).

12. Percent body fat

Percent body fat was determined by the underwater weighing technique as described by Sloan (1962). A modified formula (Brozek et al., 1973) was used in the calculation (Appendix G).

13. Percent carbohydrate, fat and protein intake

Subjects were asked to complete a computer-coded dietary record (Action, B.C., Vancouver, B.C.) for the day prior to testing. The daily diet was broken down into the percent contributions of the three main foodstuffs. Appendix H shows a sample printout.

Experimental Schedule

The following is a sample schedule of the routine followed for each testing session:

- the subject reported to the lab in the fasted state (at least 10 hours)
- the subject voided
- 300 ml of water was given
- resting blood sample was taken and fixed
- MMC calibration
- resting respiratory values were taken. The subject sat on the ergometer for at least 5 minutes while HR and respiratory values (i.e., $\dot{V}O_2$, R) leveled off
- MMC calibration
- 10 minutes of warm-up exercise. Workload was adjusted to elicit 30% $\dot{V}O_2max$
- MMC calibration
- 40 minutes of exercise. Workload was adjusted to elicit either 30, 40, 50 or 60% $\dot{V}O_2max$
- MMC calibration

- exercise blood sample was taken and fixed. The sample was taken 2 minutes post-exercise
- 30 minutes recovery. The subject sat quietly, usually reading
- recovery blood sample was taken and fixed
- urine sample was obtained

The testing order of each exercise intensity was randomized for each subject. During the ergometer work the subject was allowed to pedal at any cadence he chose. Most subjects pedalled at approximately 70-75 RPM.

Calculations

The dependent variable in this study is an adjusted non-protein R (npR'). This measure is obtained for each phase by:

- determining npR with the $\dot{V}O_2$, $\dot{V}CO_2$ and urea N_2 measures for each phase
- adjusting all npR values from a given session so that resting npR = 0.83. The result of this adjustment is a npR' value for each phase of each session.

A sample calcuation is given in Appendix I.

The adjustment to npR' values is made for two reasons. Firstly, resting R values as measured by the MMC were constantly and significantly higher than the reported range of 0.82 - 0.84 (Astrand and Rodahl, 1977; Ganong, 1979). By making the adjustment the values fall within a more acceptable physiological range. Secondly, by adjusting all resting npR values to 0.83 all subjects shared a common baseline, thus making any changes observed as a result of treatments more apparent.

Statistical Analysis

A 4 x 3 x 2 ANOVA with repeated measures on the third factor was done. Independent variables were exercise intensity (30, 40, 50 and 60% $\dot{V}O_2max$), phase (warm-up, exercise and recovery) and training state (trained and untrained), respectively. The dependent variable was npR'. Significant F's were subjected to Newman-Keul's test for multiple comparisons. Prior to any analysis a significance level of 0.05 was adopted.

A 4 x 3 x 2 ANCOVA, with similar design to the ANOVA, was also done using an intensity covariant. The covariants were generated from a regression formula (Appendix K).

Other data was tabulated and graphed. Where necessary, independent t-tests were done.

CHAPTER IV

RESULTS AND DISCUSSION

The results are presented in seven sections: physical characteristics, diet analysis, protein catabolism, work capacity measurements, exercise response measurements, covariate treatment and optimal exercise intensity. A discussion integrating the observed results concludes the chapter.

Summary tables and graphs are presented with the results. The original data and statistical analyses are located in the Appendices. Statistical significance was established at $p < 0.05$. Planned comparisons were done when F values were significant using the Newman-Keul test. Independent t-tests were performed on paired means.

Results

Physical characteristics

The weight, age and percent body fat of the subjects prior to testing are presented in Table 4.1. The only significant difference was in percent body fat, where the T group was leaner.

TABLE 4.1

Physical Characteristics of the Subjects ($\bar{x} \pm$ SD)

Group	Weight (kg)	Age (years)	Body Fat (%)
T (n = 6)	73.6 ± 8.4	22.5 ± 4.2	11.5 ± 2.6*
UNT (n = 6)	73.2 ± 4.5	24.0 ± 2.6	14.6 ± 5.0

* significant difference between T and UNT ($p < 0.05$)

The subjects verbally replied to questions pertaining to their regular physical activity over the previous two years. These responses generally indicated that the UNT group was less active than the T group.

Diet analysis

The daily diet was analyzed for average daily caloric intake and the percent contribution to this intake from CHO, fats and protein. No significant differences were found between groups in relation to the percent contribution of the three substrates. However, the T group had a significantly higher caloric intake. These data are presented in Table 4.2, along with the percent contribution for the substrates as recommended by Action B. C.

TABLE 4.2

Diet Analysis ($\bar{x} \pm SD$)

Group	Average Daily Caloric Intake (Kcal)	% CHO	% Fat	% Protein
T (n = 6)	2618 ± 797*	47.7 ± 5.1	35.5 ± 2.1	15.1 ± 2.5
UNT (n = 6)	2397 ± 430	47.6 ± 6.4	37.3 ± 7.1	14.4 ± 2.1
Action B. C. suggested intake	-	53.0	35.0	12.0

* significant difference between T and UNT ($p<0.05$)

Protein catabolism

Protein catabolism was determined for the exercise period by urinary urea N_2 analysis. There was no significant differences between the T group ($\bar{x}$ = 0.537 ± 0.244 g/hr) and the UNT group ($\bar{x}$ = 0.567 ± 0.253 g/hr).

Work capacity measurements

Three measurements were taken from the maximal ergometer test: $\dot{V}O_2max$, AerT and AnT. These data are presented in Table 4.3. Significant differences were found between groups on all three measures. While the $\dot{V}O_2max$ for the T group was higher than that of the UNT group, as designed, the UNT group showed higher thresholds when expressed as a percent of $\dot{V}O_2max$. However, when expressed in terms of absolute workload (Table 4.4), the T group showed significantly higher values.

TABLE 4.3

Work Capacity Measurements ($\bar{x} \pm SD$)

Group	$\dot{V}O_2max$ (ml/kg/min)	AerT (% $\dot{V}O_2max$)	AnT (% $\dot{V}O_2max$)
T (n = 6)	$50.5 \pm 2.3^{*}$	$50.6 \pm 5.1^{*+}$	$66.9 \pm 3.9^{*}$
UNT (n = 6)	41.1 ± 4.7	$62.4 \pm 3.8^{+}$	77.5 ± 6.4

* significant difference between T and UNT ($p < 0.05$)

\+ significant difference between AerT and AnT within groups ($p < 0.05$)

TABLE 4.4

Absolute AerT and AnT Measures ($\bar{x} \pm SD$)

Group	AerT (kpm)	AnT (kpm)
T (n = 6)	$1210 \pm 260^{*+}$	$1520 \pm 290^{*}$
UNT (n = 6)	$1080 \pm 200^{+}$	1325 ± 240

* significant difference between T and UNT ($p < 0.05$)

\+ significant difference between AerT and AnT within groups ($p < 0.05$)

Exercise response measurements

Three different measurements were taken from each test session for analysis: heart rate, lactic acid levels and non-protein R' (npR'). An analysis of variance was done on each factor with post-hoc comparisons done where F values exceeded significance ($p < 0.05$).

Heart rate was recorded during rest, warm-up, the last 30 minutes of exercise, the first 10 minutes of recovery, and the last 20 minutes of recovery. An analysis of variance revealed that there were significant phase and intensity effects, as expected. Post-hoc tests (Table 4.5) were performed to determine whether or not the relative intensities were the same for both the T and UNT group. These comparisons indicated significant differences between groups during the 40, 50 and 60% $\dot{V}O_2$max exercise intensities. This suggests that the two groups were not working at the same relative intensity. Resting heart rate also differed significantly with the T group having the lower rate. This same difference may have been reflected in the observed lower heart rate for the T group during the first 10 minutes of recovery after 30% $\dot{V}O_2$ exercise. At higher intensities the early recovery heart rate for the T groups appeared to be elevated once again.

Blood sampling for lactic acid was done at rest, immediately following exercise and after 30 minutes of recovery. An analysis of variance revealed the expected significance for intensity and phase effects. Although not significant, the lactate levels were lowered in all cases for the UNT group ($p = 0.1612$). The findings are presented in Table 4.6.

The npR' values were calculated for each phase of each testing session. These data appear in Table 4.7. An analysis of variance revealed significant F values for phase (P) main effects as well as phase and group (P x G) interaction and intensity and phase (I x P) interaction. These

Table 4.5

Heart Rates

Group	Rest+	Warm-up+	Exercise (30 min)+				Recovery (10 min)+				Recovery (20 min)+			
			30++	40	50	60	30++	40	50	60	30++	40	50	60
T (n=6)	67.3*	101.9	103.3	122.3*	139.5*	155.7*	68.2*	79.8*	83.5	98.0*	65.2	70.7	75.2	84.7
S.D.	8.3	11.3	10.6	9.4	7.1	7.8	7.3	9.5	6.5	10.5	5.5	8.2	8.0	5.0
UNT (n=6)	72.3	98.1	102.7	109.5	130.7	141.0	74.8	74.3	81.0	93.0	70.3	71.0	76.5	79.8
S.D.	7.4	7.4	7.8	13.5	10.4	11.7	6.0	6.1	10.3	12.7	7.4	6.5	9.9	10.6

* significant difference between T and UNT ($p < 0.05$)
\+ significant phase effect ($p < 0.05$)
++ significant intensity effect ($p < 0.05$)

Table 4.6

Lactic Acid (mg%)

Group	Rest*	Exercise*				Recovery*			
		30+	40	50	60	30+	40	50	60
T (n=6)	11.53	8.97	14.64	15.39	21.01	8.03	8.57	12.29	11.98
S.D.	5.76	5.53	8.99	4.63	8.16	1.10	6.49	8.65	12.10
UNT (n=6)	9.70	7.38	9.47	14.74	18.60	9.09	7.26	9.52	8.67
S.D.	4.66	3.70	3.20	4.94	7.39	4.33	0.37	3.48	3.33

* significant phase effect ($p < 0.05$)
\+ significant intensity effect ($p < 0.05$)

Table 4.7

Non-Protein R' Values

Group	Warm-up*				Exercise*				Recovery*			
	30	40	50	60	30	40	50	60	30	40	50	60
T (n=6)	1.010	1.003	0.998	1.022	1.030	1.103	1.122	1.140	0.930	0.903	0.907	0.903
S.D.	0.036	0.086	0.079	0.097	0.028	0.105	0.092	0.121	0.080	0.069	0.116	0.096
UNT (n=6)	0.940	0.915	0.968	0.962	0.975	0.997	1.068	1.052	0.893	0.898	0.910	0.935
S.D.	0.074	0.049	0.049	0.086	0.090	0.042	0.076	0.079	0.059	0.075	0.089	0.069

* significant phase effect ($p < 0.05$)

significant effects were graphed (Figures 4.1 and 4.2) and subjected to post-hoc comparative tests. Under P main effect all phases were found to be significantly different from all other phases. When the P x G interaction was tested the two groups differed significantly under the warm-up and exercise phase but not the recovery phase. The I x P post-hoc tests revealed only significant differences under the exercise phase. All exercise intensities differed from all others except the 50 and 60% $\dot{V}O_2$max intensities. Table 4.8 summarizes these post-hoc findings.

Covariate treatment

Various trends in the results, particularly in the exercise heart rate values, led to the supposition that the two groups may not have been working at the same relative intensity. For this reason analysis of covariances were used in an attempt to reduce the apparent differences between groups. This technique requires that a covariate be determined that reflected the actual relative intensity at which each subject was working. Heart rate was unacceptable because the covariate technique tended to reduce the error sum of squares, thereby increasing the group differences. The lactate values were used with similar results to the original ANOVA using npR'; the only difference being that there was no significant I x P interaction ($p = 0.1442$). Since the working intensity was suspect, a regression equation was developed to predict the intensity at which the subjects were actually working. The T group was selected as the dependent group and their exercise values for heart rate, lactate levels, and npR' were used to generate a regression equation (Appendix K). The work intensities of the UNT group were then predicted from this equation. The resulting analysis of covariance was very similar to the

Fig. 4.1. Changes in the non-protein R' (npR') value across different phases of work: warm-up, exercise and recovery. The data is presented differentiating the Trained and Untrained group (*) and with the two groups pooled (+).

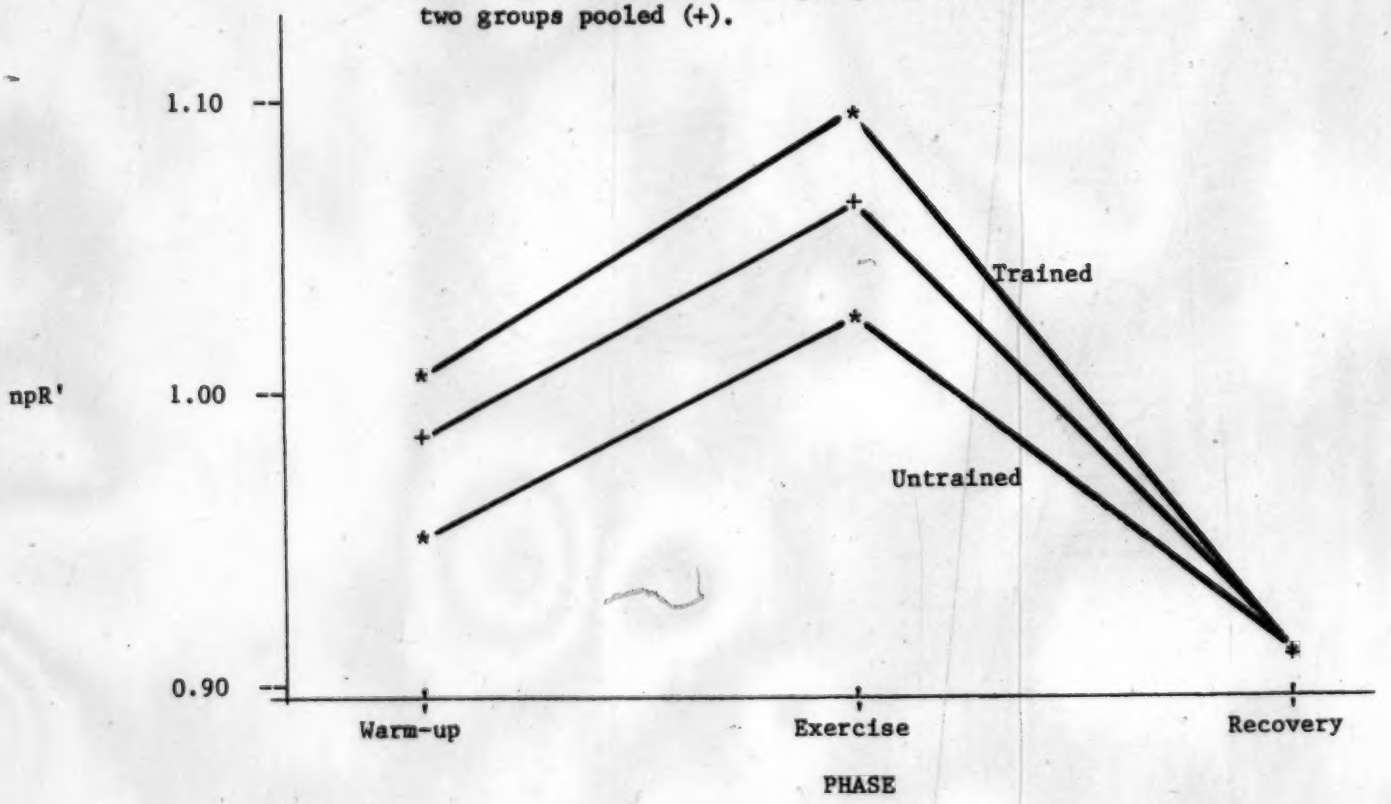


Fig. 4.2. Changes in the non-protein R' (npR') value across different phases of work: warm-up, exercise and recovery. The data is presented to differentiate the various intensities at which the subjects were working.

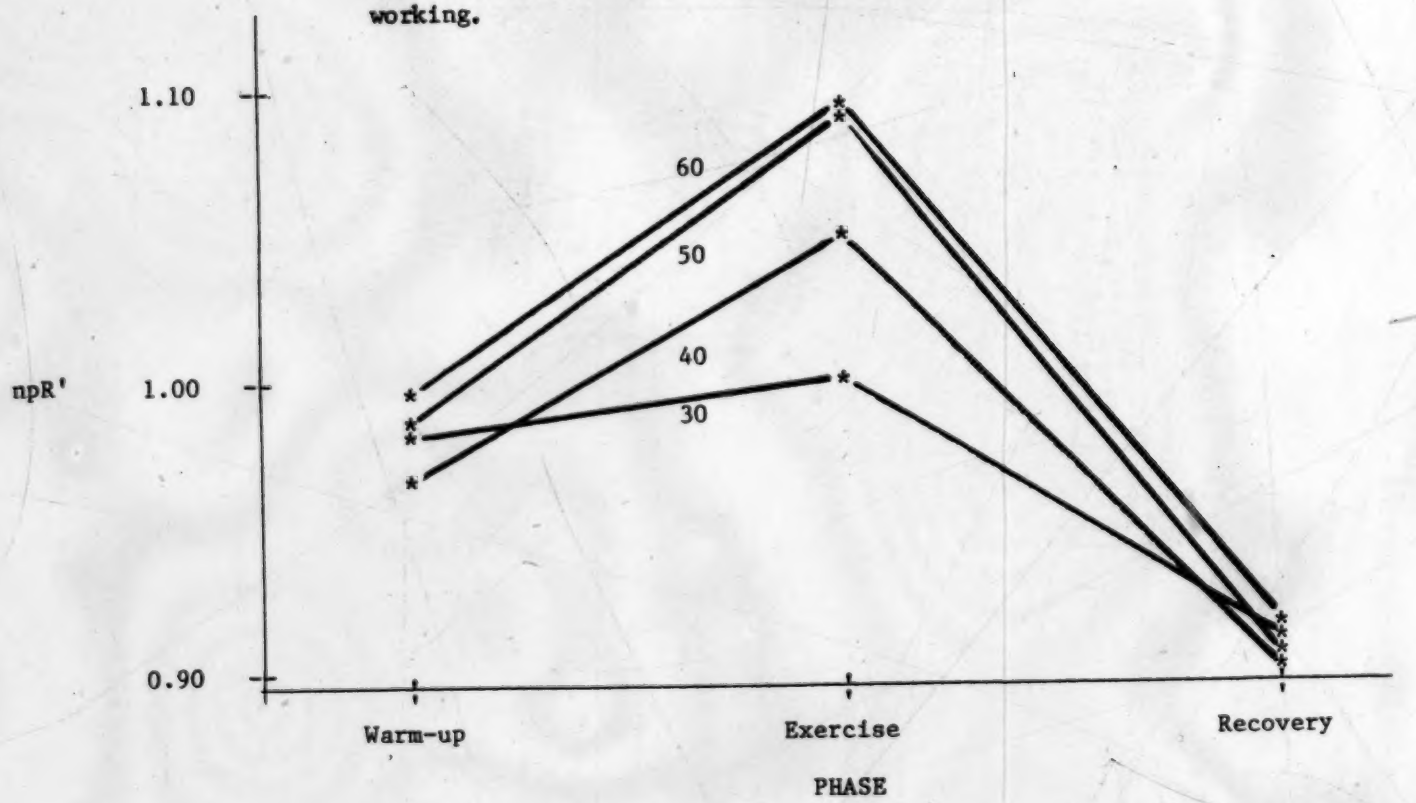


Table 4.8

Summarized Post-Hoc Comparisons From npR' ANOVA

Source	Significant F	Test	p
P	55.63		
		W vs E	0.01
		W vs R	0.05
		E vs R	0.01
P x G	3.79		
		T vs UNT, W	0.05
		, E	0.05
		, R	NS
I x P	3.89		
		W, all	NS
		E, 30 vs 40	0.05
		, 30 vs 50	0.01
		, 30 vs 60	0.01
		, 40 vs 50	0.05
		, 40 vs 60	0.05
		, 50 vs 60	NS
		R, all	NS

original ANOVA using npR'. It appeared that the intensity covariate had an effect on the G and I effects and I x G interaction but did not contribute enough to provide significance. The covariate had no effect on any other source, thus resulting in exactly the same values as the original ANOVA for P, P x G and I x P.

All ANOVA table summaries appear in Appendix L.

Optimal exercise intensity

One of the main purposes of this project was to attempt to identify, using npR' values, an exercise intensity that elicits optimal fat utilization. Unfortunately, due to the elevated npR' values (see Appendix J-VI), meaningful determination of this was not possible. Wilmore and co-workers (1976), however, established a table of R values for a wide range of work intensities. If these values are used it is possible to indirectly obtain an approximate optimal exercise intensity. The detailed procedure appears in Appendix M. The final values (Table 4.9) appear as kilocalories of energy derived during exercise from fat sources. In the case of both groups the optimal intensity appeared to be approximately 40-50% of $\dot{V}O_2max$. Since there was no significant differences found within the warm-up or recovery npR', it may be assumed that the fat utilization for the entire session was dependent only on the intensity during the exercise phase. Thus, the optimal intensity identified for the exercise phase will also be the optimal intensity for the whole session.

Discussion

Initial factors

Both the trained (T) and the untrained (UNT) groups were generally similar in physical characteristics and dietary habits. The only notable

TABLE 4.9

Kilocalories Expended From Fat Sources (kcal)

Group	Intensity (% $\dot{V}O_2$max)			
	30	40	50	60
T (n = 6)	160	200	207	185
UNT (n = 6)	133	182	205	171

difference between the groups was that the T group was significantly leaner and consumed more daily calories. In view of the reported responses to their regular physical activity, these findings were not surprising. Wood (1980) suggests that more active individuals consume more calories per day plus tend to have a lower percent body fat. Consequently, the UNT group, as a whole, might be expected to display the trends noted here. The composition of the diet, however, was similar for both groups. There did appear to be slightly more protein and less CHO consumed than recommended by Action B.C.; a trend not uncommon among North American populations (Briggs and Calloway, 1979). Several authors (Issekutz et al., 1963; Maughan et al., 1978; Pirnay et al., 1977; Benadé et al., 1973a,b) have shown shifts in R values as a result of diet manipulation. These shifts, though, were results of rather excessive manipulation. In the present case there were no such excesses in any dietary profile, so it may be concluded that the dietary factor was controlled.

There was no difference between groups with respect to protein catabolism. The normal physiological range is 0.5-1.0 g/hr (Ganong, 1979) and the two groups were within this range. As a contributor to exercise

energy, it has been reported that protein serves only a minor role (Margaria and Foa, 1939; Pirnay et al., 1977) and becomes more insignificant as the metabolic rate increases (Burztein et al., 1977). Pirnay and co-workers determined that protein accounted for 1-2% of the energy necessary to run on the treadmill for 6 hours at 50% $\dot{V}O_2$max. While some authors (Lemon and Mullin, 1980) have suggested that the protein contribution has been underestimated, it is unlikely that protein catabolism would amount to anything much more significant than is already accounted for by the determination of the non-protein R.

Aerobic and anaerobic thresholds

The AerT and AnT thresholds were determined because, according to Skinner and McLellan (1980), these transition points may have some effect on the observed substrate profile. Of particular interest is the rise in lactate levels associated with the AnT, since lactate has been shown to inhibit lipolysis (Boyd et al., 1975). The recorded AnT for the two groups was above the maximum exercise intensity for this study (see Table 4.3). Consequently, it may be assumed that the AnT did not play a significant role in the observed results. There did not appear to be any trends in the results that might be associated with the AerT. Skinner and McLellan (1980) have proposed various metabolic changes that occur at the AerT, but the techniques used in this study are either not designed to measure these changes or are too insensitive to resolve any differences.

Work intensity

Various observations have led to the supposition that the two groups were not working at the same relative intensity.

Firstly, although all subjects continued the maximal test until exhaustion, the test results were suspect. The findings that the

percentile thresholds for the UNT group were higher than those of the T group does not agree with the literature. Astrand and Rodahl (1977) note that trained individuals often have anaerobic thresholds above that of their sedentary counterparts. This conclusion is further supported by Wilmore (1977) and Wyndham (1974). These conclusions apply to both the relative (percent of $\dot{V}O_2max$) and absolute (i.e., kpm) threshold values. However, when the present data is expressed in absolute terms the threshold values for the UNT group are significantly lower. When these absolute values are compared with those reported in the literature for sedentary individuals, the UNT values appear to be reasonable (see Table 4.10), although the AnT value is higher than the reported range. It might further be assumed the threshold is a rather fixed absolute phenomenon; that is, the point at which a given person characteristically shifts from principly aerobic to anaerobic metabolism. Davis et al. (1976) has reported a significant test-retest correlation of 0.74 to support such a concept. Thus, it might be concluded that the UNT group did not reach a true $\dot{V}O_2max$ value during the maximal test. This would explain why the absolute threshold values are reasonable yet the relative ones are not. Had the UNT group as a whole achieved a higher $\dot{V}O_2max$ value, the relative threshold values would have been lowered toward, and perhaps beneath, those of the T group. The reasons for such an occurrence are not clear. It might be suggested that, as the UNT group was less accustomed to strenuous physical work, they did not push themselves as hard during the maximal test as the T group did. The result of this would be lower $\dot{V}O_2max$ values for the UNT group. Multiple maximal tests may have reduced this error.

There was further evidence to suggest that the UNT group did not achieve a true $\dot{V}O_2max$ value. At intensities of 40, 50 and 60% of $\dot{V}O_2max$,

TABLE 4.10

Comparison of UNT AerT and AnT Values to Those Reported in the Literature

Group	AerT (kpm)	AnT (kpm)	Reported Values (kpm)
UNT	1080 ± 200	1325 ± 240	610 (Davies and Sargeant, 1974)
			1098 (Wasserman et al., 1973)
			1220 (Davis et al., 1976)
			1220 (Astrand and Rodahl, 1977)
			440 (Stamford et al., 1978)
			972 (Katch et al., 1978)

the UNT group recorded significantly lower heart rates. This suggests that the UNT group was not working as hard during these relative intensities as was the T group. It is well established that trained individuals typically display lower resting submaximal and maximal heart rates (Astrand and Rodahl, 1977). If this relationship is extrapolated to relative intensity workloads, the UNT group should have a slightly elevated mean heart rate at any given relative intensity. As the reverse was true in the present case, it must be assumed that the UNT group was working at a lower intensity relative to that of the T group. This observation can be explained if it is assumed that the UNT group did not reach a true $\dot{V}O_2max$.

Although the lactate values were not significantly different, their trends support the position taken concerning the heart rates. In all cases during exercise and recovery the lactate values were lower for the UNT group (see Table 4.6). Again, it is well established that trained individuals display lower absolute lactate levels (Astrand and Rodahl,

1977). If the two groups were working at the same relative workloads, it would be assumed that the UNT group would have slightly elevated lactate levels. As the reverse was true, the previous suggestions that the UNT group did not achieve a true $\dot{V}O_2$max is supported.

Non-protein R'

The computation of the npR' value from $\dot{V}CO_2$, $\dot{V}O_2$ and urinary N_2 is a common practise (i.e., Issekutz et al., 1963; Pirnay et al., 1977). In the present study, however, the npR' values were abnormally high (see Appendix J-VI). The reason for this is not clear. The set-up and calibration instructions issued with the MMC were followed rigorously. A possible explanation is the analyzer drift over the prolonged period of analysis. Nevertheless, the attempt was made to correct for this by generating npR' values that were equated to resting values of 0.83. As the resulting npR' values still seemed abnormally high when compared to those in the literature (i.e., Wilmore et al., 1976), the npR' values have only been used qualitatively.

Substrate contribution

The npR' values obtained from the testing sessions were subjected to statistical tests. Due to the assumption that the UNT group was working at lower relative intensities, an effort was made to statistically correct for this error. Analyses of covariance were done using various covariates. Unfortunately, the best covariate (estimated intensity) did not significantly alter the ANCOVA results from those of the original ANOVA using npR'. For this reason all subsequent discussion will deal with the original ANOVA.

Significance was found for the phase (P) main effect, the phase and group (P x G) interaction and the phase and intensity (P x I) interaction.

Post-hoc analysis on the P main effect revealed that all phases were significantly different from all other phases (see Figure 4.1). In terms of the relative substrate profile fats were more prominent during recovery, less so during exercise and intermittent during warm-up. These results agree with the general observations found in the literature. In a summarizing paper, Gollnick (1978) stated that fats become less prominent as a fuel source as the intensity of work increases. The exercise phase of this study represented a higher working intensity than either the warm-up or recovery phase. Consequently the fat contribution was lower.

The recovery phase registered the lowest npR' values of all three phases. This indicates that fats were more prominent in recovery than during the other phases, although the recovery npR' value (0.91) was still greater than at that of rest (0.83). Pruett (1970) found plasma FFA levels to be elevated during recovery and suggested that they play a significant role in recovery. This position was supported by Farley and Hamley (1979) who found R to be suppressed for at least 30 minutes after 3 hours of 50% $\dot{V}O_2$max race-walking. Other supportive data includes Thompson (1980) and Haralambie and Sander (1980) who both found depressed plasma triglyceride levels during recovery, indicating enhanced lipid uptake. In the above studies, though, the recovery R reported is often below that of rest. In the present study this was not found to be the case. There may be two explanations for this. Firstly, Pruett (1970) found that the elevated recovery plasma FFA levels were dependent not on total energy expenditure but on the rate of energy expenditure (i.e., intensity as a percent of $\dot{V}O_2$max). The greater the intensity the greater and longer the duration of elevated recovery plasma FFA. This effect was most prominent at intensities of 70-80% of $\dot{V}O_2$max. In the present study the highest intensity was 60%. It is thus suggested that the intensity

was not sufficient to elicit the recovery effects observed by Pruett. The second explanation may involve the work duration. The Farley and Hamley (1979) study involved 3 hours of work while the present study only involved 50 minutes. Farley and Hamley (1979) observed a decline of 0.26 R units in 3 hours, yet only 0.06 of those occurred in the first 60 minutes. Also, after the initial 60 minutes the R value was approximately equal to the resting R value. The inference may be that exercise duration has to be of at least 60 minutes to elicit a sub-resting R value in recovery. This concept is supported by the work of Benadé and coworkers (1971, 1973a,b) who found similar substrate profiles over time.

A significant P x G interaction was found (see Figure 4.1). The direction of the difference is opposite to that that might have been hypothesized - that the T group would have lower npR' values for the work phases. Several authors (Holloszy, 1973; Holloszy, 1975, Molé et al., 1971) have reported the training adaptations to the aerobic metabolic pathways, concluding that the aerobically trained individual will have a greater oxidative potential to utilize fats. Girandola and Katch (1976) investigated the training effect on R. Their findings were that after 9 weeks of endurance training a significant decline in submaximal R was observed. These reports support the hypothesis that the T group's npR' should be lower than those of the UNT group. The discrepency is likely due to the aforementioned difficulty with the UNT group's work intensity. With the UNT group working at a lower intensity, the work npR' values would be expected to be depressed. Thus, it is not possible to determine if the T group was, in fact, more aerobically fit and oxidized more fat fuels.

A significant F was found for the P x I interaction. Post-hoc treatment determined that there were no differences within warm-up or recovery

phase while all exercise npR' differed from all other exercise npR' values, except for the 50 and 60% intensities. It was expected that no significant differences would be found during the warm-up phase as the workload (30% of $\dot{V}O_2max$) was independent of the following exercise intensity. However, it is interesting to note that the preceeding exercise intensity had no effect on the recovery npR' values. At first this result appeared to be in contradiction to those of Pruett (1970). Pruett found that working at progressively higher intensities elicited correspondingly higher plasma FFA levels during recovery; following a 50/10 minute work/rest protocol. However, these differences are not distinct until the second rest phase (i.e., 100 minutes of exercise). In the present study the exercise phase only lasted 40 minutes, conceivably not long enough to elicit the differences that Pruett found. Had the exercise phase been 2 or 3 times longer, it might be expected that the 60% intensity would elicit the lowest recovery npR' and the 30% intensity the highest.

During the exercise phase itself the npR' value increased significantly with each increment in intensity; except between the 50 and 60% levels. Such results generally agree with the literature (Pruett, 1970; Hermansen et al., 1967) that, as intensity increased, so did R. The reason for the lack of increase between the 50 and 60% levels is not clear. Pruett (1970) reported incremental increases in R through the entire intensity range from 20 to 85% of $\dot{V}O_2max$. During heavy exercise, the R value climbs progressively and may even reach 1.30 at $\dot{V}O_2max$ (DeVries, 1976). Coupled with these reports and the fact that there were no sessions done above the 60% intensity level, it is difficult to conclude that a plateau was reached in the 50-60% range.

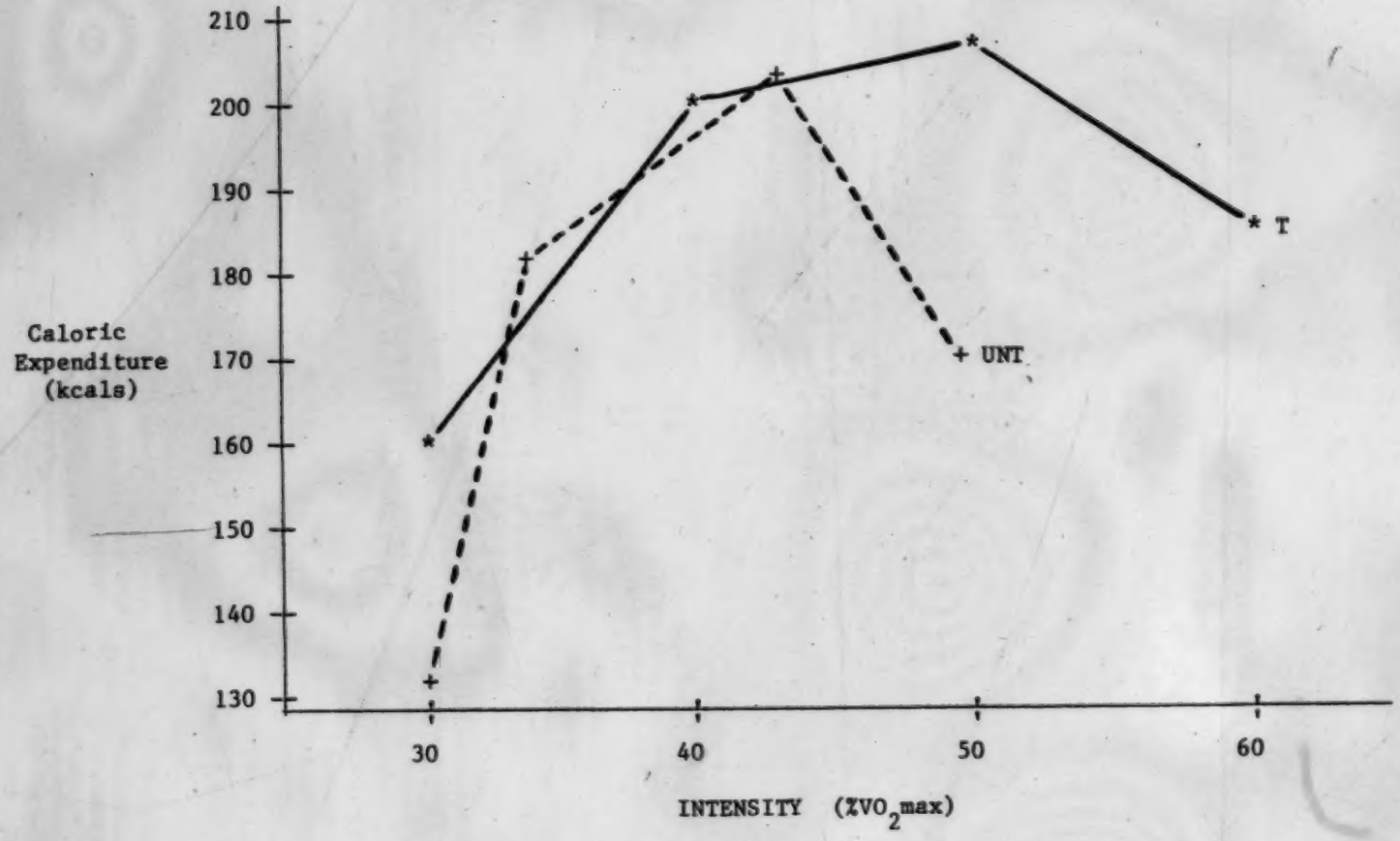
Optimal exercise intensity

An attempt was made to approximate the optimal exercise intensity for fat utilization using the exercise $\dot{V}O_2$ values from this study and the R values from another (Wilmore et al., 1976). The result was the identification of the 50% level as being optimal for both groups (see Table 4.9). However, close examination of the results revealed that, for the T group, the 40% value was only slightly lower than the 50% value (200 vs. 207 kcal). This suggests that the optimal intensity, at least for the T group, is between 40 and 50% of $\dot{V}O_2$max. Also, the UNT group is suspected of having worked at lower intensities than the T group. The regression equation suggested that the difference was 10% at the high intensity level (see Appendix K). When these regression generated intensities are graphed against the kilocalorie expenditures, the optimal intensity appears to be between 40 and 50% for both groups (Figure 4.3).

The optimal intensity determination is dependent on both the substrate percent contribution and the amount of energy expended as determined by oxygen uptake. Due to the relatively short time frame used in this particular study, the percent contribution during a given session, as determined by npR', was not expected to shift significantly. However, the recorded npR' was dependent upon the intensity during the exercise phase. Also, the energy expenditure, as measured by exercise $\dot{V}O_2$, fluctuated with exercise intensity. The combination of these two exercise intensity dependent functions resulted in optimal intensity findings.

The investigations in the literature do not, to this author's knowledge, combine a substrate contribution measure with an energy expenditure measure to determine any optimal values. Nevertheless, there are substrate studies that may help to elucidate these findings. Essén

Fig. 4.3. Kilocalorie expenditure from fat sources for Trained (*) and Untrained (+) subjects as a function of regression-generated intensity.



(1978), in reviewing the area, suggests that lipids are the prominent fuel source during work between 30 and 60% of $\dot{V}O_2$max. The author also goes on to suggest that one-half of the lipid requirement is accomplished via intramuscular stores. The differentiation between intra- and extramuscular fuel sources requires blood and biopsy techniques. As these were not done in the present study, such inferences are not possible. Also, the role of lactate has long been linked with lipid energy metabolism. Generally, lactate is thought to inhibit fat mobilization at the adipocyte (Gollnick and King, 1969; Boyd et al., 1975; Issekutz, 1965) and in the muscle (Paul and Holmes, 1975). Lactate levels do not rise appreciably above resting values until about 50% of $\dot{V}O_2$max (Astrand and Rodahl, 1977). This phenomenon was generally observed in this study (see Table 4.6). Thus, it seems reasonable that the optimal intensity level is below 50% of $\dot{V}O_2$max.

Finally, this study was designed to mimic an exercise session that might be undertaken by an individual that wishes to begin a fitness program. One of the objectives of the project was to determine if an intensity specified for cardiovascular training was also beneficial to weight loss by fat utilization. The cardiovascular training intensity, as determined by target heart rates, is usually between 60 and 85% of $\dot{V}O_2$max and maintained for more than 15-20 minutes (Astrand and Rodahl, 1977; Mathews and Fox, 1976; DeVries, 1976). The present results suggest that, for a 40 minute exercise session, the optimal intensity for fat utilization is between 40 and 50% of $\dot{V}O_2$max. However, examination of Figure 4.3 reveals that the drop-off towards the higher intensities is not extreme. There also appears to be a threshold value for cardiovascular training (60% of maximal heart rate) (Mathews and Fox, 1976). Thus, it is feasible that individuals who exercise according to the cardiovascular

training guidelines, but remain in the lower portions of the target heart rate range, will benefit from both improved cardiovascular fitness and enhanced fat utilization.

CHAPTER V

SUMMARY AND CONCLUSIONS

The fat contribution profile of 12 male subjects during submaximal exercise was studied. The subjects were split equally into trained (T) and untrained (UNT) groups, defined by $\dot{V}O_2max$. Testing sessions were done at 30, 40, 50 and 60% of the subject's $\dot{V}O_2max$ on a bicycle ergometer. Each session consisted of 4 phases: resting, 10 minutes of warm-up, 40 minutes of exercise and 30 minutes of recovery. Sessional measures included non-protein R, heart rate, venous blood for lactate and a urine analysis for protein catabolism.

It was found that the UNT group was working at lower relative workloads than the T group. This fact negated any comparison between the T and UNT group. Also, the non-protein R values were abnormally high, allowing for only qualitative discussion. There was a significant phase effect with the descending order of lipid predominance being: rest, recovery, warm-up and exercise. When the intensity factor was analysed, there appeared to be a decreasing lipid contribution with increasing intensity. No differences were found within the warm-up or recovery phase as a result of intensity. It was suggested that insufficient intensity and duration factors may have been responsible for the observed results. An attempt was made to quantify an optimal intensity for fat utilization using values reported in the literature. The result was the identification of the 40-50% $\dot{V}O_2max$ range.

Within the limitations of this study, the following conclusions appeared justified:

1. The work intensity dictated the exercise npR' value.
2. The recovery npR' did not alter with the intensity of exercise

used in this study.

3. Optimal fat utilization was during intensities between 40 and 50% $\dot{V}O_2$max.

Several recommendations for further investigations in this area can be made:

1. Test higher intensities and longer durations to evaluate the recovery phase more closely.
2. Simultaneous blood sampling and muscle biopsy techniques to validate the respiratory methods.
3. Multiple testing to ensure correct categorization of subjects into trained and untrained groups.
4. A systematic review and testing of the current reported R values and measurement techniques to validate the use of the classic substrate contribution tables.
5. The interaction with the anaerobic thresholds should be examined more closely.

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APPENDIX A

Physical Activity Readiness Questionnaire (PAR-Q)

POOR COPY
COPIE DE QUALITEE INFERIEURE

PARTICIPANT IDENTIFICATION

PHYSICAL ACTIVITY READINESS QUESTIONNAIRE (PAR-Q)*
A Self-administered Questionnaire for Adults

PAR Q & YOU

PAR-Q is designed to help you help yourself. Many health benefits are associated with regular exercise, and the completion of PAR-Q is a sensible first step to take if you are planning to increase the amount of physical activity in your life.

For most people physical activity should not pose any problem or hazard. PAR-Q has been designed to identify the small number of adults for whom physical activity might be inappropriate or those who should have medical advice concerning the type of activity most suitable for them.

Common sense is your best guide in answering these few questions. Please read them carefully and check the ☑ YES or NO opposite the question if it applies to you.

YES NO

☐☐ 1. Has your doctor ever said you have heart trouble?

☐☐ 2. Do you frequently have pains in your heart and chest?

☐☐ 3. Do you often feel faint or have spells of severe dizziness?

☐☐ 4 Has a doctor ever said your blood pressure was too high?

☐☐ 5. Has your doctor ever told you that you have a bone or joint problem such as arthritis that has been aggravated by exercise, or might be made worse with exercise?

☐☐ 6. Is there a good physical reason not mentioned here why you should not follow an activity program even if you wanted to?

☐☐ 7. Are you over age 65 and not accustomed to vigorous exercise?

If You Answered

YES to one or more questions

If you have not recently done so, consult with your personal physician by telephone or in person BEFORE increasing your physical activity and/or taking a fitness test. Tell him what questions you answered YES on PAR-Q, or show him your copy.

programs

After medical evaluation, seek advice from your physician as to your suitability for:

- unrestricted physical activity, probably on a gradually increasing basis.
- restricted or supervised activity to meet your specific needs, at least on an initial basis. Check in your community for special programs or services.

NO to all questions

If you answered PAR-Q accurately, you have reasonable assurance of your present suitability for:

- A GRADUATED EXERCISE PROGRAM - A gradual increase in proper exercise promotes good fitness development while minimizing or eliminating discomfort.
- AN EXERCISE TEST - Simple tests of fitness (such as the Canadian Home Fitness Test) or more complex types may be undertaken if you so desire.

postpone:

If you have a temporary minor illness, such as a common cold.

* Developed by the British Columbia Ministry of Health. Conceptualized and refined by the Multidisciplinary Advisory Board on Exercise (MABE). Translation, reproduction and use in its entirety is encouraged. Modifications by written permission only. Not to be used for commercial advertising in order to solicit business from the public.
Reference: PAR-Q Validation Report, British Columbia Ministry of Health, May, 1978
* Produced by the British Columbia Ministry of Health and the Department of National Health & Welfare

APPENDIX B

Informed Consent Form

CONSENT FORM

Trained ____
Untrained ____

I, _____ _____, authorize David Mann of the University of Alberta, Faculty of Graduate Studies, to administer a series of tests to determine substrate utilization during exercise.

I understand that I will complete a submaximal bicycle test during which heart rate and blood pressure will be monitored. A few days after this test I will complete a maximal aerobic power bicycle test during which respiratory measures and heart rate will be recorded. However, if I am a trained individual and used to strenuous exercise, I understand that I will not complete the submaximal test but commence with the maximal test directly. During this initial testing period I will also have my percent body fat determined via underwater weighing, regardless of whether I am trained or untrained.

It is my understanding that, after my maximal oxygen uptake has been determined, I will complete 4 sessions on the bicycle at 30, 40, 50 and 60% of this determined maximal value. The tests will commence no sooner than 3 days after the maximal test. I understand that any food that I consumed the day prior to these tests, I will record and submit before testing. Following the test I will also supply a urine sample. During these tests I understand that 3 venous blood samples will be drown from my arm by an individual trained in such techniques.

I understand that all procedures will be conducted such as to cause me as little risk and discomfort as possible. I realize that I may discontinue my participation in this study at any time I wish, without being obliged to give reasons for doing so. I further understand that the test(s) will be stopped at any time if I experience any unusual, abnormal or distressful symptoms. Finally, I understand that any questions I may have pertaining to the procedures will be answered to my satisfaction and that all information pertaining to myself will be kept confidential.

In agreeing to such tests, I waive any legal recourse against my examiner or the University of Alberta, from any and all claims resulting from personal injuries sustained or death resulting from these tests. This waiver shall be binding upon my heirs and my personal representatives.

Signature _____ Date _____

APPENDIX C

Calculations Completed by the
Beckman Metabolic Cart

Data Collected

The MMC collected the following data for further computation:

F_eCO_2	mixed expired CO_2 (fraction)
F_eO_2	mixed expired O_2 (fraction)
Temp	temperature of expired gas as it passes through the volume transducer (°C)
P_B	barometric pressure (mmHg)
Vol	cumulative expired volume (litres, ATPS)
Time	duration of measurement interval (seconds)

Body weight is entered into the computer in kilograms.

Calculations Performed

The MMC completed the following calculations using the preceding input data:

Minute volume ($\dot{V}_e$, ml/min)

1. $\dot{V}_e(\text{BTPS}) = \text{Vol} \times \frac{60}{\text{Time}} \times \frac{P_B - 25}{P_B - 47} \times \frac{273^\circ + 37^\circ C}{273 + \text{Temp}} \times 1000$

2. $\dot{V}_e(\text{STPD}) = \dot{V}_e(\text{BTPS}) \times \frac{P_B - 47}{760\ \text{mmHg}} \times \frac{273^\circ C}{310^\circ C}$

Oxygen consumption ($\dot{V}O_2$, ml/min)

3. $F_iN_2 = 1 - F_iO_2$

4. $F_eN_2 = 1 - F_eO_2 - F_eCO_2$

5. $\dot{V}_i(\text{STPD}) = \dot{V}_e(\text{STPD}) \times \frac{F_eN_2}{F_iN_2}$

6. $\dot{V}O_2 = \left(\dot{V}_i(\text{STPD}) \times F_iO_2\right) - \left(\dot{V}_e(\text{STPD}) \times F_eO_2\right)$

Substituting 3 and 4 into 5, and then 5 into 6,

7. $\dot{V}O_2 = \left[\dot{V}_e(\text{STPD}) \times \frac{(1 - F_eO_2 - F_eCO_2) \times F_iO_2}{1 - F_iO_2}\right] - \left(\dot{V}_e(\text{STPD}) \times F_eO_2\right)$

$F_iO_2 = 0.2094$, and 7 is factored,

8. $\dot{V}O_2 = \dot{V}_e(STPD) \left\{\left[0.2649 \times (1 - F_eO_2 - F_eCO_2)\right] - F_eO_2\right\}$

Oxygen uptake expressed relative to body weight (ml/kg·min) is calculated by dividing 8 by the body weight in kilograms:

9. $\dot{V}O_2(ml/kg \cdot min) = \dfrac{\dot{V}O_2(ml/min)}{\text{weight in kilograms}}$

Carbon dioxide production ($\dot{V}CO_2$, ml/min)

10. $\dot{V}CO_2 = \left(\dot{V}_e(STPD) \times F_eCO_2\right) - \left(\dot{V}_i(STPD) \times F_iCO_2\right)$

for low concentrations of inspired CO_2 $\left(\dot{V}_e(STPD) \times F_iCO_2\right) - \left(\dot{V}_i(STPD) \times F_iCO_2\right)$ is very small and $\dot{V}_i(STPD)$ is a close approximation of $\dot{V}_e(STPD)$ for $F_iCO_2 = 0.0003$ (atmospheric air). Therefore:

11. $\dot{V}CO_2 = \dot{V}_e(STPD) \times (F_eCO_2 - 0.0003)$

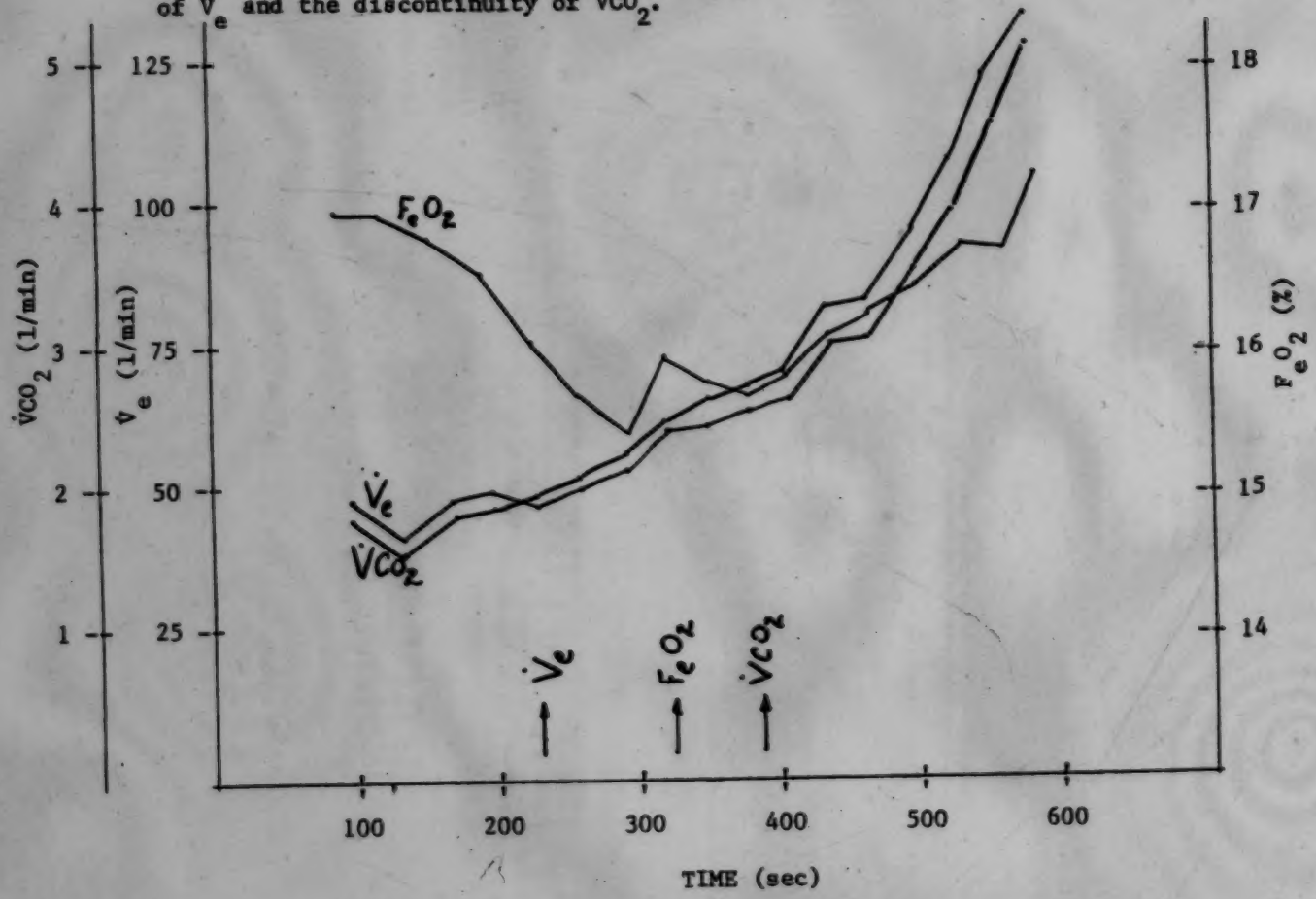
Respiratory quotient

12. $R = \dfrac{\dot{V}CO_2(ml/min)}{\dot{V}O_2(ml/min)}$

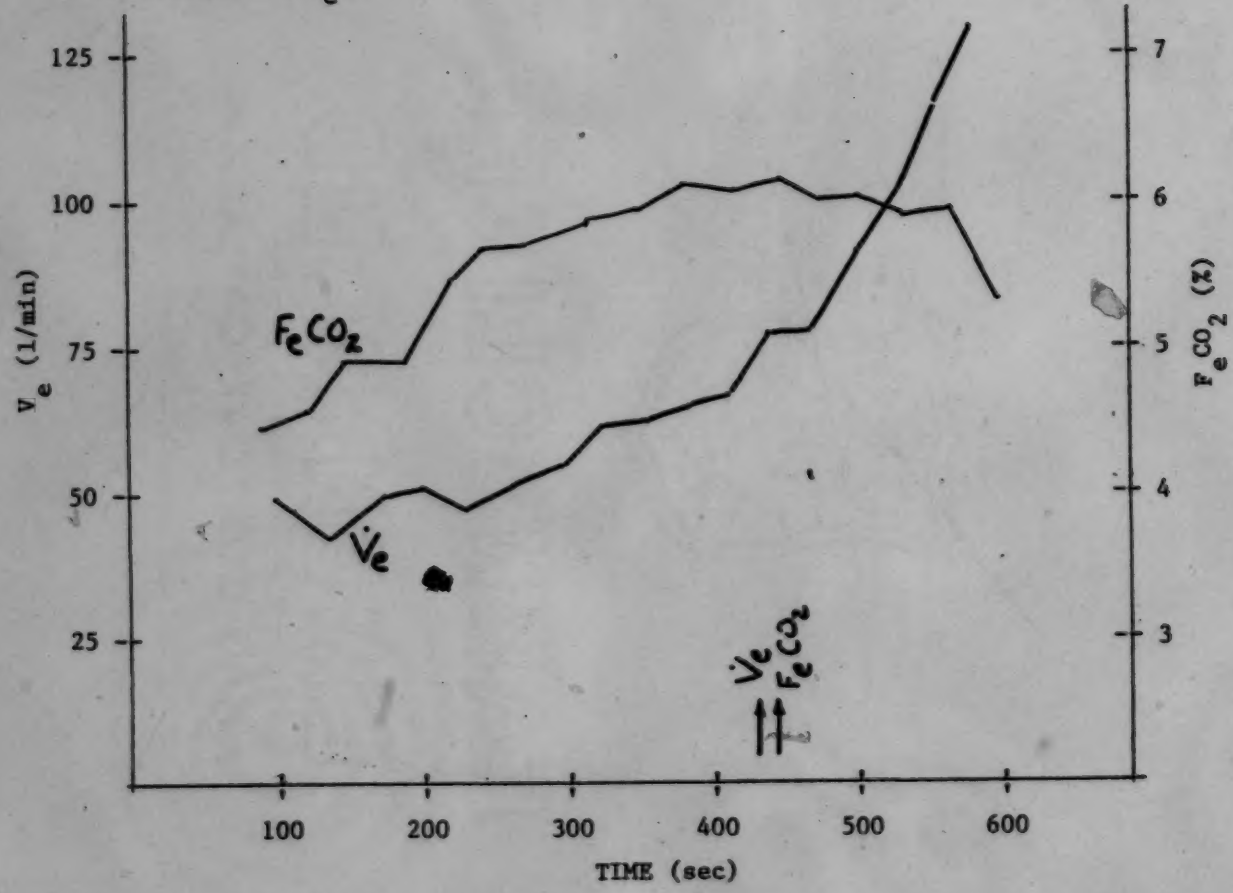
APPENDIX D

Graphical Determination of
Aerobic and Anaerobic Thresholds

Aerobic Threshold (AerT) Determination. The point is determined by the averaged values of the minimum F_eO_2, the first discontinuity of V_e and the discontinuity of VCO_2.



Anaerobic Threshold (AnT) Determination. The point is determined by the averaged values of the maximum F_eCO_2 and the second discontinuity of $\dot{V}_e$.



APPENDIX E

Lactic Acid Determination

Collection and storage

The subject voided just prior to testing. He was then given 300 ml of cool water to drink. Urine samples were collected immediately after the completion of the resting phase.

The sample volume was recorded and approximately 3 ml was transferred to a test tube. This was covered and frozen at -57°C until urea N_2 analysis.

Analysis

The analysis was done with a Sigma Chemical Co. Urea Nitrogen kit (No. 640) for use with spectrophotometer. The kit included phenol nitroprusside, alkaline hypochlorite, urase solution and urea nitrogen standard solution. Double assays were done on each sample.

Procedure

1. Test tubes are labelled BLANK, STANDARD and as many TEST as necessary.
2. Add 0.5 ml urase solution to each test tube.
3. To BLANK: add 0.010 ml DH_2O
   To STANDARD: add 0.010 ml urea nitrogen standard solution
   To TEST: dilute urine sample by 1000. Add 0.010 ml diluted urine.
   Mix and incubate 5-10 min at 37°C.
4. To all test tubes add, in the following order:

   1.0 ml phenol nitroprusside solution
   1.0 ml alkaline hypochloride solution
   5.0 ml DH_2O
5. Incubate at room temperature for 20-30 min.
6. Read STANDARD and TEST against BLANK at 570 nm.

Calculations

$$\text{Urea } N_2 \text{ (mg \%)} = \frac{A_{570}\ \text{TEST}}{A_{570}\ \text{STANDARD}} \times 3000$$

$$\text{Urea } N_2 \text{ (g/hr)} = \frac{A_{570}\ \text{TEST} \times \text{Vol(ml)}}{A_{570}\ \text{STANDARD} \times \text{Time (hrs)}} \times 0.03$$

APPENDIX F

Urea Nitrogen Determination

Collection, fixation and storage

Whole blood was removed using the vacutube method. One millilitre of blood was pipetted into 4 ml of cold (0°C) 4% perchloric acid and mixed. The test tube was covered and stored at 0°C for less than 90 minutes.

The stored blood-perchlorate mixture was spun at 3000 RPM for 10 minutes. The supernatent was pipetted off and frozen at -57°C until lactic acid analysis. The pellet was discarded.

Analysis

The analysis was done with a Sigma Chemical Co. Pyruvate Acid and Lactic Acid Determination kit (No. 726-UV and 826-UV) for use with spectrophotometer. The kit included nicotinamide adenine dinucleotide (NAD) vials, glycine buffer and lactic dehydrogenase (LDH). Double assays were done on each sample.

Procedure

1. One vial is used for every 2 assays.
2. Into each vial pipette:

   2.0 ml glycine buffer
   4.0 ml DH_2O
   0.1 ml LDH

   Cap the vial and invert to mix.
3. Contents of all vials are combined into a flask.
4. Into labelled test tubes pipette 2.8 ml of solution from step 3.
5. Add 0.2 ml of 4% perchlorate to BLANK.
6. Add 0.2 ml sample solution to labelled test tubes.
7. Incubate for 30 min at 37°C.
8. Read against BLANK at 340 nm.

Calculations

Blood lactic acid (mg %) = $A_{340} \times 130.2$.

APPENDIX G

Calculation of Percent Body Fat

TABLE 5.2 - CALCULATIONS FOR UNDERWATER WEIGHING TECHNIQUE

MEASUREMENTS: SUBJECT: _____

(1) Wt. in air _____ (lbs.)

(2) Vital capacity (V.C.) _____ (litres) x 61.02 = _____(cu.in.)

(3) Residual Volume 30% V.C. = _____(cu.in.)

(4) Vol. Gastro-intestinal track (VGI) = 7.01 (cu.in.)

(5) Wt. in water (full inspiration) = _____(lbs.)

Wt. in water = $\left[\frac{\text{Chart Reading x belt wt.}}{75}\right]$ - belt weight (lbs)

= _____ (must be negative)

CALCULATIONS:

(6) Total body air (T.B.A.) = V.C. _____(cu.in.) (from 2 above)

+ R.V. _____(cu.in.) (from 3 above)

+ RGI 7.01 (cu.in.)

= _____ x 0.0362 = (lbs.)

(7) True wt. in water = weight in water (from 5 above) _____(lbs.)

+ total body air (from 6 above) _____(lbs.)

= _____(lbs.)

(8) Body Volume = wt. in air (1) _____ - true wt. in water (7) _____ = _____(lbs.)

(9) Body density = wt. in air (1) _____ body volume (8) _____ X density of H_2O _____ = _____

(10) % Fat = $\left[\frac{4.570}{\text{Body Density}} - 4.142\right]$ x 100

= _____%

(11) Lbs. fat = [_____ (% fat) x _____ (wt. in air)] - 100

= _____(lbs.)

(12) Lbs. fat free wt. = _____ wt. in air (1)

- Lbs. fat (11) _____ = _____ (lbs. fat free wt.)

APPENDIX H

Sample Diet Analysis

21/ 2/80 HELLO . HERE IS YOUR PERSONALIZED NUTRITION EVALUATION BASED ON THE INFORMATION YOU REPORTED.

CANADIANS ARE GENERALLY EATING POORLY, ACCORDING TO STUDIES DONE BY NUTRITION CANADA AND OTHERS. HERE IS AN OPPORTUNITY FOR YOU TO EVALUATE YOUR OWN DIET AND THEREBY GET A BETTER UNDERSTANDING OF THE AMOUNT AND VARIETY OF FOODS YOU NEED TO MAINTAIN GOOD HEALTH.

YOUR PERSONAL DATA

THE PERSONAL DATA YOU REPORTED IS SHOWN BELOW. THE COMPUTER HAS IDENTIFIED THE IDEAL WEIGHT RANGE FOR A PERSON OF YOUR AGE AND SEX.

- MALE . BIRTH DATE 1/10/57. AGE 22. HEIGHT 177 CM (70 INS.),
- WEIGHT 70 KG (154 LBS.), MEDIUM FRAME SIZE
- THE AVERAGE WEIGHT RANGE FOR YOU IS 67 TO 74 KG (150 TO 165 LBS.)

YOUR ACTIVITY DATA

THE ACTIVITY DATA YOU REPORTED HAS BEEN AVERAGED. THE CALORIE EQUIVALENT FOR AN AVERAGE PERSON IS SHOWN FOR THE DIFFERENT ACTIVITY LEVELS.

- LEVEL 1	7.43 HOURS RESTING	REQUIRES	534 CALORIES -
- LEVEL 2	10.06 HOURS SEDENTARY	REQUIRES	1042 CALORIES -
- LEVEL 3	5.07 HOURS LIGHT ACTIVITY	REQUIRES	973 CALORIES -
- LEVEL 4	0.64 HOURS ACTIVE	REQUIRES	216 CALORIES -
-	TOTAL CALORIC EXPENDITURE =		2765 CALORIES -

CALORIES ARE A UNIT OF MEASURE FOR ENERGY IN THE SAME WAY THAT INCHES ARE A MEASURE FOR LENGTH. YOUR SIZE, BOTH HEIGHT AND WEIGHT, AND YOUR ACTIVITY LEVEL DETERMINE HOW MANY CALORIES YOU USE EACH DAY. IF YOU PROVIDE YOUR BODY WITH THE SAME NUMBER OF CALORIES THAT YOU USE UP YOUR WEIGHT WILL REMAIN CONSTANT. WEIGHT GAIN OCCURS WHEN THE BODY HAS TO STORE EXCESS CALORIES (UNUSED ENERGY) AND WEIGHT LOSS OCCURS WHEN THE BODY HAS TO USE STORED ENERGY.

YOUR CALORIE BREAKDOWN

YOUR CALORIES COME FROM THE FOLLOWING SOURCES, AS SHOWN IN THE TABLE BELOW, AND ARE EXPRESSED AS AN APPROXIMATE PERCENTAGE OF YOUR TOTAL CALORIC INTAKE. THE IDEAL BALANCE FOR CALORIE SOURCES IS GIVEN.

	CARBOHYDRATE	FAT +	PROTEIN
- YOURS	44.0	35.4	19.0 -
- IDEAL	53.0	35.0	12.0 -

+ THE FEDERAL MINISTRY OF HEALTH RECOMMENDS THAT FAT INTAKE BE NO MORE THAN 35% OF YOUR TOTAL CALORIES TO HELP PREVENT HEART DISEASE. REFER TO 'GOOD EATING TO GUARD YOUR HEART' AVAILABLE FROM YOUR LOCAL HEALTH UNIT.

YOUR FOOD GROUPS

THE FOOD AND DRINK ITEMS YOU REPORTED HAVE BEEN CATEGORIZED INTO THE SIX BASIC FOOD GROUPS. THE FOLLOWING TABLE COMPARES THE NUMBER OF SERVINGS YOU NEED DAILY WITH THE AVERAGE NUMBER OF SERVINGS IN YOUR DIET.

FOOD GROUPS	NO. OF RECOMM. SERVINGS	AVERAGE NUMBER OF SERVINGS YOU HAD
1. GRAINS, BREADS & CEREALS	3.0 TO 5.0	7.1
2. MILK AND MILK PRODUCTS	2.0 TO 2.0	2.6
3. MEAT AND ALTERNATIVES	2.0 TO 2.0	3.3
4. FRUIT AND VEGETABLES	4.0 TO 5.0	6.1
5. FAT AND OILS		5.9
6. SWEETS AND DESSERTS		4.7

YOUR NUTRIENT BREAKDOWN

THE FOOD AND DRINK ITEMS YOU REPORTED HAVE BEEN SEPARATED INTO THE FOOD COMPONENTS SHOWN BELOW. THE RECOMMENDED AMOUNTS FOR WEIGHT MAINTENANCE FOR A PERSON OF YOUR SEX, AGE AND ACTIVITY ARE COMPARED TO YOUR INTAKE.

FOOD OR NUTRIENT	UNIT	RECOMMENDED AMOUNT	YOUR INTAKE AMOUNT	% OF RECOM.	INTAKE LESS THAN RECOM.
CALORIES	KCAL	2730.0	2436.4	89	YES
PROTEIN	GM	56.0	115.8	206	
THIAMIN	MG	1.5	1.4	91	YES
NIACIN	MG	20.0	47.7	238	
RIBOFLAVIN	MG	1.8	2.9	159	
VIT. B6	MG	2.0	1.8	87	YES
FOLATE	MCG	200.0	208.9	104	
VIT. B12	MCG	3.0	5.2	172	
VIT. C	MG	30.0	146.6	488	
VIT. A	RE	1000.0	1133.6	113	
CALCIUM	MG	800.0	1303.8	162	
PHOSPHORUS	MG	800.0	1902.0	237	
IRON	MG	10.0	15.7	157	
PANTOTHENIC	**MG	5.0	5.3		
SODIUM	**GM	2 TO 8	2.7		
FIBRE	**GM	5 TO 8	5.6		
CARBOHYDRATE	GM		260.1		
FAT	GM		95.9		
ALCOHOL	GM		10.9		

** SUGGESTED VALUES AS NO STANDARDS HAVE BEEN SET

IT IS USUAL FOR YOUR NUTRIENT INTAKE TO VARY FROM DAY TO DAY. SOME ARE STORED AND ONLY REQUIRE AN ADEQUATE WEEKLY INTAKE, SUCH AS IRON AND VITAMIN A. OTHERS, LIKE VITAMIN C, ARE NOT STORED AND ARE NEEDED DAILY. IT IS BEST TO HAVE AN ADEQUATE SUPPLY OF ALL NUTRIENTS ON A DAILY BASIS.

YOUR DAILY NUTRIENT BREAKDOWN

FOOD OR NUTRIENT	UNIT	DAY #1 AMOUNT	DAY #1 %	DAY #2 AMOUNT	DAY #2 %	DAY #3 AMOUNT	DAY #3 %	DAY #4 AMOUNT	DAY #4 %
CALORIES	KCAL	2407.0	91	2285.1	83	2865.8	104	2680.6	98
PROTEIN	GM	120.8	229	110.9	212	123.4	220	154.6	293
THIAMIN	MG	1.6	109	1.4	90	1.3	86	1.1	74
NIACIN	MG	51.2	255	52.4	261	45.1	225	72.1	360
RIBOFLAVIN	MG	3.9	210	2.2	120	3.2	176	3.1	170
VIT. B6	MG	2.0	99	0.8	39	1.0	51	2.1	106
FOLATE	MCG	203.7	101	177.0	88	198.4	99	195.3	97
VIT. B12	MCG	5.0	167	2.2	73	3.5	116	3.6	118
VIT. C	MG	146.8	489	118.6	395	120.9	403	116.8	389
VIT. A	RE	648.9	64	1220.5	122	1254.7	125	669.1	66
CALCIUM	MG	1690.1	211	671.4	83	1726.2	215	1101.7	137
PHOSPHORUS	MG	2467.4	308	1580.1	197	2111.8	263	2271.1	283
IRON	MG	17.1	170	16.7	166	14.6	145	17.3	172
PANTOTHENIC**	MG	7.9		3.9		4.8		5.9	
SODIUM	**GM	3.7		1.8		2.8		3.2	
FIBRE	**GM	6.1		3.9		4.1		5.0	
CARBOHYDRATE	GM	250.5		201.9		263.1		231.4	
FAT	GM	91.7		111.5		137.2		102.3	
ALCOHOL	GM	26.8		0.0		13.4		26.8	

YOUR DAILY INTAKE BY FOOD GROUPS IN SERVINGS PER DAY

FOOD GROUPS	DAY #1	DAY #2	DAY #3	DAY #4
GRAINS,BREAD,ETC	7.70	8.00	8.00	5.00
MILK & MILK PROD	3.50	1.00	4.50	2.00
MEAT & ALTERNATE	3.45	4.25	2.70	5.60
FRUIT & VEGETABL	7.75	4.50	3.75	7.00
FATS AND OILS	4.50	7.00	10.00	4.50
SWEETS & DESSERT	2.00	0.00	5.00	0.00

YOUR DAILY ACTIVITY

ACTIVITY	DAY #1	DAY #2	DAY #3	DAY #4
INACTIVE	8.00	7.00	8.00	7.00
NOT VERY ACTIVE	13.00	14.00	6.00	12.00
SLIGHTLY ACTIVE	3.00	2.50	9.00	5.00
ACTIVE	0.00	0.50	1.00	0.00

RECOMMENDATIONS

** YOU HAVE EATEN FEWER CALORIES THAN YOU ARE USING UP THROUGH ACTIVITY. THIS WILL CAUSE YOU TO LOSE WEIGHT IF CONTINUED.

** WE SUGGEST YOU CHOOSE MORE FOODS CONTAINING CALORIES

** WE SUGGEST YOU CHOOSE MORE FOODS CONTAINING THIAMIN

** WE SUGGEST YOU CHOOSE MORE FOODS CONTAINING VITAMIN B6

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** PROTEINS ARE MADE UP OF A NUMBER OF AMINO ACIDS. THE AMINO ACIDS THAT CANNOT BE SYNTHESIZED IN THE BODY MUST ALL BE PRESENT IN YOUR DIET ON A DAILY BASIS BECAUSE THEY ARE REQUIRED FOR THE GROWTH OF NEW TISSUE, REPAIR OF OLD TISSUE AND REGULATION OF IMPORTANT BODY FUNCTIONS. THE DEMAND FOR ENERGY TAKES FIRST PRIORITY IN METABOLISM. IF CARBOHYDRATE AND FAT ARE NOT CONSUMED IN SUFFICIENT AMOUNTS SOME OF THE AMINO ACIDS WILL BE USED AS A SOURCE OF ENERGY AND WILL NOT BE AVAILABLE FOR THE SYNTHESIS OF BODY PROTEINS. WHEN ENERGY CONSUMPTION IS LOW PROTEIN IS USED LESS EFFICIENTLY. THE CORRECT BALANCE BETWEEN PROTEIN, FAT AND CARBOHYDRATE INTAKE IS VERY IMPORTANT. THE RECOMMENDATION FOR PROTEIN INTAKE IS CLOSE TO DOUBLE THE AVERAGE REQUIREMENT FOR SOMEONE YOUR AGE AND WEIGHT TO INSURE THAT VARIATIONS IN INDIVIDUAL NEEDS ARE MET.

** CARBOHYDRATES SUPPLY THE MOST EFFICIENT SOURCE OF ENERGY FOR YOUR BODY. MOST OF THE CARBOHYDRATES IN YOUR DIET COME FROM STARCHES AND SUGARS. IT IS HEALTHIER TO CONSUME MOST OF YOUR CARBOHYDRATES AS STARCHES BECAUSE THESE FOODS ALSO CONTAIN MANY NECESSARY VITAMINS AND MINERALS AS WELL AS FIBER. CEREALS OR WHOLE GRAINS, LEGUMES, FRUITS AND VEGETABLES ARE NOURISHING SOURCES OF CARBOHYDRATES. THAT PORTION OF YOUR DIET WHICH COMES FROM SUGARS IS NOTED IN FOOD GROUP 6. A SERVING SIZE IS EQUAL TO 1 TEASPOON OF SUGAR AND IS APPROXIMATELY 15 NON-NOURISHING CALORIES. THE FOODS YOU HAVE EATEN THAT ARE PARTICULARLY HIGH IN SUGAR ARE NOTED IN THE FOOD INPUT LIST.

** FATS SUPPLY THE MOST CONCENTRATED SOURCE OF ENERGY FOR YOUR BODY AND ARE REQUIRED AS A SOURCE OF ESSENTIAL FATTY ACIDS, PARTICULARLY LINOLEIC ACID, AND AS A CARRIER OF THE FAT SOLUBLE VITAMINS A, D, E AND K. THE TYPE AND AMOUNT OF FAT YOU EAT IS IMPORTANT TO YOUR HEALTH AND MANY HEALTH PROFESSIONALS ENCOURAGE A MINIMUM INTAKE OF FAT FROM ANIMAL FOOD SOURCES BALANCED BY SOME FAT FROM VEGETABLE FOOD SOURCES. THAT PORTION OF YOUR DIET WHICH COMES FROM FATS IS NOTED IN FOOD GROUP 5. EACH UNIT IS EQUAL TO THE FAT CONTAINED IN 1 TEASPOON OF BUTTER AND CONTAINS APPROXIMATELY 45 CALORIES. FOR MORE INFORMATION PLEASE REFER TO "GOOD EATING TO GUARD YOUR HEART".

** VITAMINS AND MINERALS, GENERALLY SPEAKING, ARE SPECIAL SUBSTANCES THAT ARE NEEDED IN SMALL AMOUNTS BY YOUR BODY TO PERFORM COMPLEX CHEMICAL REACTIONS THAT ARE VITAL TO ITS PROPER FUNCTIONING AND HEALTH. VITAMINS PLAY THEIR MOST IMPORTANT ROLE BY INSURING THAT OTHER NUTRIENTS ARE USED EFFECTIVELY. MINERALS ACT AS BODY REGULATORS AND AS BUILDING MATERIALS FOR BOTH HARD AND SOFT TISSUES. THE PAMPHLET "FUNCTIONS AND SOURCES OF NUTRIENTS IN FOODS", THAT COMES WITH THIS PRINTOUT, IS MORE SPECIFIC. NUTRIENTS ARE INTERRELATED AND THE PROPER BALANCE MUST BE MAINTAINED. EXCESS, AS WELL AS DEFICIENCY, OF ANY NUTRIENT MAY BE HARMFUL. IN OTHER WORDS, A CERTAIN AMOUNT OF EACH NUTRIENT IS ESSENTIAL FOR GROWTH AND MAINTENANCE OF HEALTH; TOO LITTLE CAN CAUSE DEFICIENCY DISEASES AND TOO MUCH CAN PRODUCE TOXICITY OR METABOLIC DISTURBANCES. IT IS IMPORTANT TO KEEP THIS IN MIND BEFORE SELF PRESCRIBING ANY VITAMIN, MINERAL OR OTHER NUTRIENT SUPPLEMENT. A DAILY DIET THAT CONTAINS A VARIETY OF DIFFERENT FOODS FROM WITHIN EACH OF THE IMPORTANT FOOD GROUPS IS MOST LIKELY TO GIVE YOU THE BEST BALANCE OF ALL NUTRIENTS.

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FOOD INPUT LIST

DAY 1

AMOUNT	SERVING SIZE	FOOD NAME	COMMENT
3.50	1 CUP, 8 OZ	MILK-2%	VITAMIN D SOURCE
4.00	1 SLICE	BREAD,100% WHOLE GRAIN	
1.00	1 CUP	CEREAL,DRY ENRI.,FLAKED	
2.00	2	CRACKERS,WHOLE WHEAT	
1.00	1	MUFFIN,WHOLE GRAIN,BRAN	
1.00	1/2 CUP	FRUIT COCKTAIL,CND.,SW.	HIGH SUGAR CONTENT
1.00	1/2	GRAPEFRUIT	
1.00	1	ORANGE/TANGERINE	
0.25	1/2 MEDIUM	CUCUMBER	
1.00	2 LARGE	LETTUCE LEAVES	
0.50	1 OZ	MEATS,DELI TYPE	HIGH FAT CONTENT
1.00	1 TBSP	MAYONNAISE	HIGH FAT CONTENT
2.00	1 BOT.,12 OZ	BEER	HIGH CALORIC CONTENT
1.00	1 CUP	TEA	
1.00	1 CUP	CHICKEN A LA KING	RECIPE VARIES
1.00	1 CUP	CHOP SUEY & MEAT	
1.00	1 CUP	CHOW MEIN,NO NOODLES	

DAY 2

AMOUNT	SERVING SIZE	FOOD NAME	COMMENT
1.00	1 CUP, 8 OZ	MILK-2%	VITAMIN D SOURCE
4.00	1 SLICE	BREAD,WHITE ENRICHED	
2.00	2	CRACKERS,RUSK/MELBA	
1.00	1	MUFFIN,WHOLE GRAIN,BRAN	
0.50	1 CUP	MACARONI,PASTA,CKD.	
1.00	1 MEDIUM	APPLE	
2.00	1/2 CUP	ORANGE JUICE,UNSW.	
1.00	1/2 CUP	CORN,KERNEL	
1.00	2 LARGE	LETTUCE LEAVES	
1.50	1	EGG,PLAIN,CKD.	
2.00	1 SLICE	BACON,SIDE	HIGH FAT CONTENT
1.00	2 OZ	HAM/BACKBACON	HIGH FAT CONTENT
3.00	3 OZ	CHICKEN,BAKED	
1.00	1 TSP	MARGARINE	HIGH FAT CONTENT
1.00	1 TBSP	MAYONNAISE	HIGH FAT CONTENT
2.50	1 CUP	COFFEE	
1.00	1 CUP	TEA	

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DAY 3

AMOUNT	SERVING SIZE	FOOD NAME	COMMENT
2.50	1 OZ	CHEESE,CHEDDAR,HARD	
2.50	1 CUP, 8 OZ	MILK-2%	VITAMIN D SOURCE
3.00	1 SLICE	BREAD,100% WHOLE GRAIN	
3.00	2	CRACKERS,RUSK/MELBA	
1.00	1 CUP	MACARONI,PASTA,CKD.	
1.00	1 MEDIUM	APPLE	
2.00	1/2 CUP	ORANGE JUICE,UNSW.	
1.50	2 LARGE	LETTUCE LEAVES	
1.00	1	EGG,PLAIN,CKD.	
2.00	1 OZ	MEATS,DELI TYPE	HIGH FAT CONTENT
2.00	3 OZ	CHICKEN,BAKED	
1.00	1 TSP	MARGARINE	HIGH FAT CONTENT
3.00	1 TBSP	MAYONNAISE	HIGH FAT CONTENT
1.00	1 BOT.,12 OZ	BEER	HIGH CALORIC CONTENT
1.00	1 CUP	TEA	
1.00	1 TBSP	JAM/JELLY/HONEY/SYRUP	HIGH SUGAR CONTENT
1.00	1 IN. SQUARE	CAKE,FRUIT	HIGH SUGAR AND FAT

DAY 4

AMOUNT	SERVING SIZE	FOOD NAME	COMMENT
2.00	1 CUP, 8 OZ	MILK-2%	VITAMIN D SOURCE
2.00	1 SLICE	BREAD,100% WHOLE GRAIN	
2.00	2	CRACKERS,RUSK/MELBA	
1.00	1 MEDIUM	APPLE	
1.00	1 MEDIUM	BANANA	
1.00	1/2 CUP	ORANGE JUICE,UNSW.	
2.00	3 OZ	CHICKEN,FRIED	
0.50	2 TBSP	PEANUT BUTTER	FAT & INCOMPLETE PROTEIN
1.00	1 TSP	MARGARINE	HIGH FAT CONTENT
2.00	1 BOT.,12 OZ	BEER	HIGH CALORIC CONTENT
1.00	1 CUP	COFFEE	
1.00	2	COOKIE,OAT MEAL	HIGH CALORIC CONTENT
1.00	1 CUP	CHICKEN A LA KING	RECIPE VARIES
1.00	1 CUP	CHOP SUEY & MEAT	
1.00	1 CUP	CHOW MEIN,NO NOODLES	

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PLEASE NOTE: THE ABOVE COMMENTS REFER TO EACH FOOD IN A GENERAL SENSE. IF YOUR DIET IS IN NEED OF SOME CHANGE, THESE COMMENTS SHOULD HELP YOU TO DECIDE WHICH FOODS TO AVOID - IF YOUR DIET IS FINE, CONSIDER THEM AS "INFORMATION ONLY".

A REMINDER: THE ANALYSIS PRESENTED ABOVE IS BASED ON STANDARDS FOR NORMALLY HEALTHY CANADIAN CHILDREN AND ADULTS. IF YOU ARE WORRIED ABOUT YOUR DIET WE RECOMMEND THAT YOU SEEK HELP FROM A PROFESSIONAL DIETITIAN-NUTRITIONIST, YOUR LOCAL HEALTH DEPARTMENT OR YOUR PHYSICIAN.

WE HAVE TRIED TO INDICATE THE CHARACTERISTICS OF YOUR DIET YOU MIGHT LIKE TO CONSIDER ADJUSTING SO THAT YOUR BODY CAN FUNCTION AT ITS BEST. WE WISH YOU GOOD HEALTH.

COMPUTER PROGRAM DEVELOPED FOR ACTION B.C.

NUTRIENT ANALYSIS BASED ON CURRENT CANADIAN DIETARY STANDARDS.

APPENDIX I

Calculations for Determining npR'

1. Raw data needed:

   - $VO_2(\ell)$ and $VCO_2(\ell)$ for each phase of rest, warm-up, exercise and recovery
   - urea N_2 production (g/hr) over the entire session

2. The npR value must be computed for each phase. The amount of O_2 and CO_2 associated with protein catabolism is subtracted according to:

   1 g urea N_2 = 5.91 ℓ O_2 consumed

   1 g urea N_2 = 4.76 ℓ CO_2 produced

   (Hawk, 1951)

   The resulting formula to determine npR for each phase is:

$$npR = \frac{\dot{V}CO_2(1) - \left(4.76\ \ell/g \times \frac{\text{phase time (min)}}{60\ \text{min/hr}} \times N_2(g/hr)\right)}{VO_2(1) - \left(\frac{5.91\ \ell/g \times \text{phase time (min)}}{60\ \text{min/hr}} \times N_2(g/hr)\right)}$$

3. The phase npR are adjusted to normal R measures. The resting npR is adjusted to 0.83 as a reference and all subsequent phases are adjusted the same amount in the same direction. The result is a modified npR (npR') for each phase.

   For example:

Phase	npR	Δ	npR'
Rest	0.91	0.08	0.83*
Warm-up	0.94	0.08	0.86
Exercise	0.99	0.08	0.91
Recovery	0.93	0.08	0.85

*reference value for rest (0.83)

APPENDIX J

Raw Data

APPENDIX J

Raw Data

Appendix I.	Subject Characteristics
Appendix II.	Aerobic and Anaerobic Thresholds
Appendix III.	Protein Catabolism
Appendix IV.	Lactic Acid
Appendix V.	Mean Heart Rate
Appendix VI.	npR Values Before Transformation to npR'

J-I Subject Characteristics

Group	Subject No.	Age (yrs)	Weight (Kg)	Body Fat (%)	Max. O_2 Uptake (VO_2max) (ml^2O_2/Kg/min)
Trained (T)	1	28	87.9	10.6	47.7
	2	19	73.0	14.0	50.1
	3	23	71.8	12.3	48.5
	4	20	67.0	6.9	53.5
	5	27	77.5	13.5	50.4
	6	18	64.3	11.7	52.7
Untrained (UNT)	7	26	68.2	10.0	43.6
	8	27	76.3	11.3	44.0
	9	25	77.1	12.0	42.4
	10	24	73.4	23.1	33.1
	11	22	67.2	13.1	46.6
	12	20	77.2	18.0	43.1

J-II Aerobic and Anaerobic Thresholds

Group	Subj. No.	AerT (ml/Kg/min) V_e	F_eCO_2	VCO_2	$\bar{x}$	$\%VO_2max$ (%)	AnT (ml/Kg/min) V_e	F_eCO_2	$\bar{x}$	$\%VO_2max$ (%)
Trained (T)	1	-	25.5	25.0	25.2	52.8	35.0	26.7	30.9	64.7
	2	26.4	26.2	26.0	26.2	52.3	37.0	33.5	35.3	70.4
	3	20.0	20.0	19.0	19.8	40.8	38.5	30.5	34.5	71.1
	4	25.2	31.5	32.4	29.7	55.5	35.0	37.5	36.3	67.8
	5	18.0	27.0	30.4	25.1	49.9	30.4	30.7	30.5	60.5
	6	25.0	32.7	25.0	27.6	52.3	35.4	35.2	35.3	67.0
Untrained (UNT)	7	27.0	25.0	27.1	26.4	60.5	36.0	30.5	33.3	76.3
	8	22.5	28.3	-	25.4	57.7	44.0	33.1	38.6	87.6
	9	27.5	26.8	31.3	28.5	67.3	34.1	29.3	31.7	74.8
	10	18.0	24.2	20.0	20.7	62.6	26.0	-	26.0	78.6
	11	29.7	31.0	32.0	30.9	66.3	35.0	39.2	37.1	79.6
	12	26.7	23.5	27.5	25.9	60.1	32.0	26.7	29.4	68.1

J-III Protein Catabolism (g/hr)

Group	Subject No.	Intensity (%VO_2max)				
		30	40	50	60	mean ± S.D.
Trained (T)	1	0.913	0.670	1.157	0.668	0.852 ± 0.234
	2	1.093	0.809	0.641	0.407	0.738 ± 0.289
	3	0.314	0.371	0.367	0.295	0.338 ± 0.038
	4	0.306	0.390	0.405	0.622	0.431 ± 0.135
	5	0.460	0.507	0.442	0.318	0.432 ± 0.081
	6	0.441	0.337	0.403	0.554	0.434 ± 0.091
Untrained (UNT)	7	0.400	0.275	0.192	0.457	0.331 ± 0.120
	8	0.620	1.248	0.615	0.759	0.811 ± 0.300
	9	0.283	0.734	0.454	0.660	0.533 ± 0.204
	10	0.942	0.698	0.495	0.881	0.754 ± 0.201
	11	0.441	0.494	0.419	0.820	0.544 ± 0.187
	12	0.487	0.463	0.636	0.144	0.433 ± 0.207
						0.567 ± 0.253

J-IV Lactic Acid (mg%)

Group	Subj. No.	Intensity (%VO_2max) 30 R	30 E	30 R	40 R	40 E	40 R	50 R	50 E	50 R	60 R	60 E	60 R
Trained (T)	1	9.77	7.36	6.84	9.51	12.89	2.93	5.23	13.28	8.85	10.68	28.45	2.60
	2	10.35	-	-	1.17	9.77	0.00	-	10.22	-	4.69	10.42	3.97
	3	11.33	9.05	9.11	15.62	8.92	9.77	7.10	13.87	8.92	18.88	30.21	14.06
	4	-	18.29	-	8.53	32.68	20.01	14.78	23.96	-	15.43	23.83	10.81
	5	12.95	6.19	7.36	11.33	12.89	11.20	7.81	15.36	6.25	8.01	12.50	5.40
	6	12.82	3.09	8.79	29.23	10.68	9.11	16.54	15.62	25.13	11.91	20.64	35.02
Untrained (UNT)	7	13.02	11.46	10.68	8.20	8.72	7.29	8.21	15.82	10.32	4.82	10.94	4.75
	8	15.95	-	10.03	7.68	7.94	7.55	5.92	11.33	4.04	12.37	21.61	8.85
	9	13.87	4.10	6.38	15.69	14.78	7.03	21.22	15.04	10.22	9.51	12.30	9.57
	10	-	-	-	7.42	-	-	16.54	23.70	13.41	7.16	20.77	8.72
	11	-	9.70	14.84	8.46	6.32	6.77	6.25	9.77	12.24	6.05	15.17	5.86
	12	15.66	4.49	3.51	9.31	9.57	7.68	8.33	12.76	6.90	1.76	30.80	14.26

J-V Mean Heart Rates

Group	Subj. No.	Rest				Warm-up				Last 30 min. Exercise				First 10 min. Recovery				Last 20 min. Recovery			
		30	40	50	60	30	40	50	60	30	40	50	60	30	40	50	60	30	40	50	60
Trained (T)	1	66	68	69	63	100	98	107	100	104	128	140	160	68	83	89	96	65	75	81	82
	2	75	72	60	66	108	106	94	105	109	126	128	157	79	83	80	97	69	75	66	88
	3	69	65	71	66	109	93	106	101	107	106	149	162	69	78	87	111	66	66	81	89
	4	72	68	64	92	104	100	126	121	109	125	138	163	72	90	80	108	65	71	73	88
	5	55	62	59	58	83	83	93	79	82	117	138	145	54	62	74	82	55	57	66	76
	6	82	76	57	61	105	118	108	98	109	132	144	147	72	83	91	94	71	80	84	85
Untrained (UNT)	7	69	72	65	65	85	91	84	91	90	98	115	126	71	71	73	76	66	69	74	75
	8	69	62	64	52	94	94	90	93	99	94	121	141	73	72	67	87	65	64	64	73
	9	76	77	70	67	105	104	106	86	106	131	147	144	68	69	82	95	64	68	72	68
	10	75	75	82	73	106	100	197	100	112	109	139	139	85	83	97	105	83	82	94	93
	11	78	81	78	70	100	96	102	104	101	107	133	138	74	70	82	91	69	68	77	77
	12	79	74	83	80	106	105	102	104	108	118	129	158	78	81	85	106	75	75	78	93

J-VI npR Values Before Transformation to npR'

Group	Subj. No.	Rest	Intensity ($\%VO_2max$) 30 W	30 E	30 R	40 W	40 E	40 R	50 W	50 E	50 R	60 W	60 E	60 R
Trained (T)	1	1.19	1.41	1.41	1.18	1.32	1.45	1.10	1.51	1.62	1.40	1.26	1.42	1.08
	2	1.03	1.20	1.27	1.26	1.17	1.28	1.12	1.27	1.47	1.31	1.39	1.55	1.28
	3	0.90	1.06	1.08	0.98	1.02	1.12	1.05	0.99	1.08	0.84	1.05	1.15	0.94
	4	1.00	1.15	1.17	1.13	1.13	1.17	1.06	1.081	1.17	1.05	1.07	1.14	0.93
	5	1.00	1.13	1.11	0.97	1.35	1.45	1.09	1.38	1.42	1.15	1.22	1.33	1.08
	6	1.06	1.20	1.23	1.14	1.14	1.27	1.11	1.16	1.27	1.09	1.27	1.38	1.24
Untrained (UNT)	7	1.02	1.16	1.25	1.09	1.05	1.09	0.93	1.26	1.39	1.24	1.32	1.41	1.30
	8	1.08	1.10	1.14	1.10	1.10	1.17	1.24	1.17	1.29	1.15	1.45	1.50	1.34
	9	0.94	1.10	1.22	1.01	1.09	1.26	1.03	0.98	1.11	0.95	0.98	1.11	0.97
	10	1.04	1.06	1.06	1.12	1.43	1.46	1.23	0.94	1.00	0.97	0.94	1.04	0.94
	11	0.95	1.16	1.14	1.03	1.14	1.20	1.07	1.17	1.28	1.08	0.89	0.94	0.85
	12	1.11	1.26	1.24	1.21	1.12	1.21	1.10	1.26	1.33	1.27	1.21	1.33	1.21

APPENDIX K

Regression Procedures for Covariant Determination

A regression equation was generated to predict exercise intensity from exercise HR, lactate levels and npR'. The equation was made using the data from the T group:

Int = 0.43(HR) + 0.11(LA) + 21.65(R) - 35.97

The multiple R was 0.9356 (R^2 = .8753).

This equation was used to generate intensity values for both the T and UNT groups at each session. These values were then used as covariants in an ANCOVA.

Group	Intensity (% $\dot{V}O_2$max)			
	30	40	50	60
T	31.8	41.3	50.2	58.2
UNT	30.2	33.8	43.8	49.3

APPENDIX L

ANOVA Tables

Table L-I

Summary Table of F ratios From 3-Way ANOVA Using Heart Rate

Source	F
G	0.22
P	31.51**
PxG	2.59
I	85.54**
IxG	2.15
PxI	168.98**
PxIxG	2.31*

* $p < 0.05$
** $p < 0.01$

Table L-II

Summary Table of F Ratios From 3-Way ANOVA Using Lactic Acid

Source	F
G	2.45
P	2.72
PxG	0.07
I	3.43*
IxG	1.19
PxG	6.25**
PxIxG	0.53

* $p < 0.05$
** $p < 0.01$

Table L-III

Summary Table of F Ratios From 3-Way ANOVA Using npR'

Source	F
G	2.35
I	1.66
IxG	0.35
P	55.63**
PxG	3.79*
IxP	3.89**
IxPxG	1.00

* $p < 0.05$
** $p < 0.01$

Table L-IV

Summary Table of F Ratios From 3-Way ANCOVA Using npR' and Intensity (covar.)

Source	F
G	2.20
Covar	0.42
I	0.15
IxG	0.34
Covar	0.03
P	55.63**
PxG	3.79*
Covar	0.00
IxP	3.89**
IxPxG	1.00
Covar	0.00

* $p < 0.05$ ** $p < 0.01$

APPENDIX M

Indirect Determination of Optimal Exercise Intensity

Wilmore et al. (1976) arranged their table of R values in ascending METS (2 - 16), with 1 MET = 3.5 ml O_2/kg/min. The group's (T and UNT) average oxygen uptake for the four exercise intensities was determined and equated to a Wilmore R value. Subsequent manipulation to obtain non-protein values showed only slight changes, so the original Wilmore values were used throughout the following computations.

Wilmore R Values

Group	Intensity (% $\dot{V}O_2$max)			
	30	40	50	60
T	0.79	0.80	0.83	0.87
UNT	0.79	0.78	0.80	0.83

From the tables of Lusk (1924), the percent contribution corresponding to each R value is obtained.

The final step is to determine the energy contribution by fats at each intensity. This value is generated by multiplying together the percent contribution, the average litres of oxygen consumed and the approximate caloric equivalent of 5 kcal/ℓ O_2 (Mathews and Fox, 1976). For example, the trained group under 30% $\dot{V}O_2$max:

$$0.717 \times 44.70\ \ell\ O_2 \times 5\ \text{kcal}/\ell\ O_2 = 160\ \text{kcal}$$